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# The Journal of Thoracic Surgery

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# The Journal of Thoracic Surgery

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# The Journal of Thoracic Surgery

VOL. 5 AUGUST, 1936 NO. 6


## Original Communications

### SPECIAL TRAINING FOR THORACIC SURGERY*


CARL EGGERS, M.D.
NEW YORK, N. Y.


THE purpose underlying the founding of the American Association for Thoracic Surgery was to solve the problems connected with the development of surgery of the chest. Men representing associated specialties were invited to join. As a result of this the membership of our organization during the early years of its existence included surgeons, internists, phthisiologists, radiologists, physiologists, anesthetists, and endoscopists.

Such a group, working in the interest of a surgical specialty, is unique among surgical societies. It was their cooperation which was largely responsible for the rapid development of our association.

Soon after its organization the American Association for Thoracic Surgery considered it advantageous to publish its transactions as a separate report in order to bring the progress made during the preceding year to the attention of the medical profession. Beginning with the 1923 issue of the *Archives of Surgery* a special supplement on thoracic surgery was published annually. Eventually there was an increasing desire among the members to have a Journal of their own, and this wish was realized with the publication of the first issue of the JOURNAL OF THORACIC SURGERY, October, 1931.

Members of our Association have made valuable contributions to the diagnosis of intrathoracic lesions. They have helped solve the problems connected with the respiratory and circulatory difficulties encountered in an open pneumothorax, the proper selection of cases for operation, methods of anesthesia, surgical approach, drainage, and after care. They have also contributed much important experimental research. All these, combined with the actual improvement in surgical technic, and the courageous and often daring approach to difficult surgical problems

*Presidental address before the Nineteenth Annual Meeting of the American Association for Thoracic Surgery at Rochester, Minnesota, May 4, 5, 6, 1936.

have attracted much attention and have fired the enthusiasm of many of the younger men in the profession. One may safely say that thoracic surgery, though the most recent among the surgical specialties, is today the most spectacular, and is making progress more rapidly than any other branch of surgery. Much as this may be applauded, there is a certain danger inherent in this unless it is properly controlled. One may well ask the question, Is it not time for taking stock to determine where we are going? Are our efforts directed along the proper channels; is everything being done to educate the general profession on matters pertaining to thoracic surgery; are the men doing thoracic surgery properly trained and qualified; are there sufficient safeguards to our work, or should we, the thoracic surgeons, seek more counsel from associated medical men, especially the internists, phthisiologists, research workers, and others?

There are those among us who feel that the time has come for thoracic surgery to be designated a separate specialty. Their reasoning is based on the facts that in thoracic surgery, more than in other branches of surgery, physiologic problems connected with respiration and circulation play an important rôle and have to be studied and understood. They further are of the opinion that it is necessary to be familiar with medical affections of the intrathoracic organs, their diagnosis and medical as well as surgical treatment, that a thoracic surgeon should be conversant with all the various diagnostic methods, such as the study of vital capacity, bronchography, pneumothorax, endoscopy, and those which may still be developed. They also believe that he should be able to perform all the modern therapeutic nonoperative procedures carried out through an endoscope, including the use of radium, electrocoagulation, etc., that he should know the subject of pneumothorax, its indications and contraindications, intrapleural pneumolysis, all the procedures used in the diagnosis and treatment of esophageal conditions, etc., and that in addition he should be a good general surgeon.

On the other hand, there are others who feel that thoracic surgery as such is safest in the hands of the general surgeon with a special interest in thoracic surgery. They contend that thoracic surgery has been developed by the general surgeon and that progress in this field is likely to be safer and better if continued that way. They advocate close cooperation with medical men, radiologists, and endoscopists on the theory that the ultimate aim is better served in that way.

The subject that has probably contributed most to the general discussion of where thoracic surgery is leading us, is pulmonary tuberculosis. Inasmuch as it is a surgical disease under certain conditions, the proper care of these cases has developed into a problem. In many instances operations have been performed by men inadequately trained for these extensive interventions with results that leave much to be desired. One should be fair in one's criticism, however; for it is not altogether by design, but rather the result of suddenly opening up a

large field for surgical practice without previous conception of the task and without proper preparation for its management. The treatment of pulmonary tuberculosis formerly consisted, and to a large degree still does, of physical and mental rest, fresh air and sunshine, nourishing food and appropriate medication. To make such treatment available, patients were taken from the city to sanatoriums located out of town, and that is where most of them were when the wave of surgical enthusiasm swept the country. How to make it possible for these patients to enjoy the benefits of surgery was a problem. Most sanatoriums were not equipped to do even minor surgery, much less the extensive procedures frequently required. It was not only the operation, but the difficulty of furnishing the important nursing and after care. Some solved it by moving all those patients requiring major operations to a well-equipped general hospital, others by establishing surgical facilities at the sanatorium. In the latter case a surgeon was called from the city to perform the operation, or arrangements were made to have a permanent resident surgeon.

In order to get their opinion on these important controversial points the following questionnaire was sent to our entire membership. We wanted to determine who constitutes our society, which specialties are represented, what training have the members had, and what are their views concerning the necessary educational qualifications for the practice of thoracic surgery.

**QUESTIONNAIRE**

1. Are you a general surgeon with a special liking or inclination for thoracic surgery?

or

2. Are you limiting your practice to thoracic surgery?

or

3. Are you an internist or *phthisiologist?*

or

4. Are you a radiologist?

or

5. Are you an endoscopist?
6. Have you acquired your knowledge of thoracic surgery or of diseases of the thoracic organs in the course of your practice?

or

7. Have you had special training in this subject, such as a fellowship or residency?
8. If so, how long a time have you spent in special preparation?
9. Do you consider it advantageous or necessary to have special training for the practice of thoracic surgery?

or

10. Do you believe it is preferable or sufficient to acquire the necessary proficiency in thoracic surgery as a part of the training for general surgery?
11. Do you believe thoracic surgery should be developed as a definite specialty?

or

12. Do you believe thoracic surgery should be practiced as a part of general surgery?

---

Signature

It is interesting to note that of the 169 to whom questionnaires were mailed, 159 or 94 per cent responded. On the basis of this return our members may be classified as follows:

| | |
|---|---|
| General surgeons | 97 or 61.0% |
| Thoracic surgeons | 18 or 11.3% |
| Internists and phthisiologists | 32 or 20.0% |
| Radiologists | 3 or 1.9% |
| Endoscopists | 7 or 4.4% |
| Retired | 2 or 1.3% |

ANSWERS TO SOME OF THE QUESTIONS

Question 7: How many had special training.

| | |
|---|---|
| Of the 97 general surgeons | 34 or 35.1% |
| Of the 18 thoracic surgeons | 15 or 83.3% |

Question 9: How many believe it is advantageous or necessary to have special training for the practice of thoracic surgery.

| | |
|---|---|
| Of the 97 general surgeons | 91 or 93.9% |
| Of the 18 thoracic surgeons | 18 or 100.0% |
| Of the 44 nonsurgical members | 39 or 88.7% |

Question 10: How many believe it is preferable or sufficient to acquire the necessary proficiency in thoracic surgery as a part of the training for general surgery.

| | |
|---|---|
| Of the 97 general surgeons | 59 or 60.8% |
| Of the 18 thoracic surgeons | 3 or 16.7% |
| Of the 44 nonsurgical members | 13 or 29.5% |

Question 11: How many believe thoracic surgery should be developed as a definite specialty.

| | |
|---|---|
| Of the 97 general surgeons | 30 or 30.9% |
| Of the 18 thoracic surgeons | 17 or 94.5% |
| Of the 44 nonsurgical members | 28 or 63.7% |

Question 12: How many believe thoracic surgery should be practiced as a part of general surgery.

| | |
|---|---|
| Of the 97 general surgeons | 84 or 86.7% |
| Of the 18 thoracic surgeons | 3 or 16.7% |
| Of the 44 nonsurgical members | 23 or 52.2% |

An analysis of the questionnaire shows that 115 or 72.3 per cent are surgeons, of whom the great majority are general surgeons with a special interest in thoracic surgery. They are either teachers at universities or attending surgeons in general hospitals. Their work brings them in contact with all types of patients, including thoracic cases. They understand and cope with affections of the chest wall and of the pleura, inflammatory conditions and tumors of the lungs, affections of the mediastinum, pericardium, heart, esophagus, and diaphgram. The gradual development of thoracic surgery as a specialty to its present

position may be attributed to these general surgeons who have paid particular attention to this work. One may assume therefore that further development will depend on their continued interest. On the other hand, one may take the attitude that thoracic surgery has become so specialized that it should be segregated from general surgery. In some of the large centers or large general hospitals such an attitude may be justified and may bring good results. Whether we are ready for it is doubtful. In smaller hospitals in large cities and in the hospitals of smaller communities the general surgeon will probably have to continue to do thoracic surgery. Because of the fascinating subject, the general interest attaching to it, and the demand for men qualified to do thoracic surgery, one may reasonably expect that many of the younger general surgeons will aim to improve themselves in this field.

Of the 97 general surgeons especially interested in thoracic surgery 79 stated they had acquired their experience in this type of work in the course of their practice. Several attributed their interest and proficiency partly to war experiences with chest injuries and diseases. Only a few had taken special training. When it came to the question of whether they considered it advantageous or necessary to have special training in preparation for the practice of thoracic surgery, an overwhelming majority, 91 or 93.9 per cent, expressed themselves strongly in favor of it, while only 6 thought such special training unnecessary. To question 10, asking whether they believe it is preferable or sufficient to acquire the necessary proficiency in thoracic surgery as a part of the training for general surgery, 59 answered in the affirmative and 32 in the negative. Several of those answering in the affirmative supplemented their answer by stipulating that the necessary training can be had only if there is sufficient thoracic surgery connected with general surgery. To the question whether thoracic surgery should be developed as a definite specialty, 29 answered yes and 68 no. When asked whether thoracic surgery should be practiced as a part of general surgery, 83 answered yes and 14 no.

The replies and comments of this group definitely indicate that the great majority of general surgeons doing thoracic surgery at the present time realize that, though they themselves advanced largely by gradually acquiring the knowledge and skill they possess today, thoracic surgery has reached a place which requires special study and application for its mastery.

A smaller group among our members classify themselves as specialists in thoracic surgery. It was rather surprising to find as many as 18 who practice thoracic surgery exclusively. This is a new development which has been made possible by the rapid evolution of this specialty. That it is growing, there seems little doubt. Whether we should force it in any way is questionable. Perhaps our attitude toward it should be one of sympathetic support to the degree of helping to provide opportunities

for all those young men who are eager to improve themselves in this type of work. That such special training will be helpful may be judged by the opportunities that have been offered properly qualified men and by the demand of younger men for this type of training. Even today many of the larger general hospitals welcome men on their general surgical staff who have had special training in thoracic surgery or who specialize in it. This does not mean that they have to give up general surgery; quite the contrary, they may be better men for keeping their interest in general surgery alive. Of the 18 thoracic surgeons limiting their practice to this specialty 15 have had special training, either a fellowship or a residency, all 18 are of the opinion that special training in this field is essential, 15 believe that the necessary proficiency cannot be acquired while training for general surgery, 17 favor the development of thoracic surgery as a definite specialty, and only 3 feel that it may well be combined with general surgery. In a general way one gets the impression that this group represents younger men, but that is not really true. Neither can one say that they represent a more progressive viewpoint, for among the surgeons of the general surgical group there are many just as young and quite as progressive. They simply have a different viewpoint which probably has been influenced by their training and environment.

It is rather dangerous to make comparisons on the relative merits of the two groups on the basis of contributions to the literature, but one may safely say that general surgeons with a special interest in thoracic surgery compete effectively when it comes to experimental research or to clinical contributions.

The viewpoints of nonsurgical members of our association are interesting for comparison and also because they look upon the subject from another angle. Though the great majority have had no systematic special training in their own field, and have gradually acquired their knowledge and proficiency until now they are experts, they almost unanimously feel that all those who want to practice thoracic surgery should have special training. Many are of the opinion that the necessary proficiency can be acquired while training for general surgery, provided there are a sufficient number of chest cases on the general service. An equal number, however, are convinced that a period of special training should be set aside. The great majority state that thoracic surgery should be developed as a definite specialty; though a fair percentage are of the opinion that it may well be combined with general surgery. Very interesting viewpoints are expressed by some of the phthisiologists who have been confronted with the problem of providing efficient surgical attention in out-of-the-way places. There is among them a strong tendency to have thoracoplasties done at the sanatoriums where the patients are surrounded by personnel which knows and understands them.

There are only a few men among our own members who are phthisiologists and who, without a general surgical training, are devoting themselves to the surgery of pulmonary tuberculosis. The idea is not new, and some of the development of this type of surgery, in Europe as well as in this country, is due to the painstaking effort and pioneering work of such men. There are, however, outside of our membership a considerable number of men who do thoracic surgery for pulmonary tuberculosis without having studied or practiced general surgery. They have simply "taken up" this kind of work because the opportunity presented, or of necessity, or because it seemed to offer lucrative remuneration. Some are phthisiologists who feel that they should do their own surgery in order to carry through the entire treatment of a patient with pulmonary tuberculosis. That the guiding motive of many of these men is an honest and sincere one, may not be doubted. Arguments have been advanced that all the work is outside of the lungs, affecting the chest wall, and that proficiency in this type of surgery may be acquired by men who are not general surgeons. One may concede that in individual instances this may be correct, but it is inadvisable to set this up as a principle.

The question whether it is safe to permit untrained or inexperienced men to perform these serious operations on patients who are suffering from a constitutional illness requires an answer. Any operation performed on these cases is likely to be more far reaching in its effects than, for instance, a general surgical operation in an otherwise healthy individual. Unforeseen surgical conditions may be encountered during operation, or serious surgical complications may develop which require judgement and skill which a trained surgeon is more likely to possess than one not so trained. All considerations of the subject lead to the conclusion that untrained surgeons should not be permitted to do the surgery of pulmonary tuberculosis.

Immediately we come to this conclusion, however, we are faced with the serious problem of how to provide surgical treatment for tuberculous patients in out-of-the-way places. There seem to be two ways of accomplishing this, either by moving all patients requiring a major operation to a general hospital where a qualified surgeon is available or by providing well-trained thoracic surgeons at the sanatoriums. It would be necessary to have them become resident surgeons so that they may become familiar with the preoperative condition of the patient and to take care of the surgical after treatment. The custom now followed in many places of having a city surgeon come out to perform the technical part of an operation on a patient he has never seen before, and abandoning the after-treatment to some one else should be condemned. The problem has by no means been solved. It is acute, and we should be seriously concerned about it.

It appears at the present time that the organization of our association is a good one, and the plan to have nonsurgical members associated with

us is beneficial. A frank exchange of ideas with them will continue to direct us along the right channels.

There are certain questions confronting us, however, which require solution. The survey of our membership shows conclusively that there is overwhelming opinion in favor of special training for those who want to engage in thoracic surgery. Whether it should be developed as a definite specialty seems to be of relatively minor importance at the present moment. That question will regulate itself as time goes on.

The important points to engage the attention of our society are to what degree special training is advisable and how to provide it. All of us are agreed that it should be on the basis of a good training in general surgery. Beyond that, different paths may be followed.

For those who want to practice thoracic surgery as a specialty, an extensive course would evidently best serve the purpose, a course which may include experimental work, training in all the diagnostic and non-operative technical procedures, as well as ample experience in the performance of all well-known thoracic operations. A period of service in a tuberculosis sanatorium may be of value.

On the other hand, for those men who want to practice thoracic surgery in conjunction with general surgery, a less extensive course may be arranged.

In addition to these, it will be well to consider seriously the question of whether it is wise to recognize a third group, and to provide systematized training for those men who do not aspire to become thoracic surgeons in the sense mentioned above, but who want to prepare themselves for the practice of surgery of pulmonary tuberculosis. The time has come for our association to make a study of these questions and to formulate a policy.

# TRAINING OF THE THORACIC SURGEON FROM THE STANDPOINT OF THE GENERAL SURGEON*

EVARTS A. GRAHAM, M.D.
ST. LOUIS, MO.

THE question under discussion brings up the ever recurring one of how far specialization should occur within a specialty. The practice of surgery itself is, of course, a specialty, but the rapid growth of certain aspects of surgery has already led to the inevitable result that specialists in those fields have arisen. This fact has been given more or less general recognition by the recent creation of independent American boards for the qualification of specialists in such branches of surgery as ophthalmology, otolaryngology, gynecology, orthopedic surgery, and urology. The chief question which concerns us is really whether or not the field of thoracic surgery should be regarded as another branch of surgery of a sort which might require special qualification like that which has arisen in the other branches of surgery just mentioned. Other questions which arise are those of how much training in general surgery the thoracic surgeon should have and also whether or not the thoracic surgeon should limit himself exclusively to that branch of surgery.

There can be no argument, it seems to me, over the fact that no matter to what special field one wishes to limit his work a sound training in the general aspects of medicine in its broad sense is essential. We recognize that fact in compelling, for example, a prospective ophthalmologist to go through in his medical school work a survey of all of the various branches of medicine. The only exception to this rule is in the field of dentistry in which, in spite of a smattering of general anatomy, physiology, pathology, etc., the student's training is almost entirely concentrated on the particular specialty for which he expects to prepare himself. Likewise, in the various surgical specialties, with the exception perhaps of ophthalmology and of otolaryngology, it has become more and more recognized that an experience in general surgery is helpful in the education of the specialist. Certainly any one who undertakes the practice of the more difficult sort of thoracic surgery must be thoroughly acquainted with the fundamental principles of surgery, those, for example, which deal with shock, hemorrhage, infection, etc. It would seem also that countless other situations must arise in which a considerable experience in general surgery would be most helpful. The thoracic surgeon moreover must be acquainted with those features of physiology which are peculiar to his special field, such as the im-

*Read at the Nineteenth Annual Meeting of the American Association for Thoracic Surgery at Rochester, Minnesota, May 4, 5, 6, 1936.

portance of altered intrathoracic pressure, to name only one. It would seem inconceivable that any one could jump into major thoracic surgical work by any short cut which would eliminate a sound training in general surgery over a period of years.

The question of specialization in thoracic surgery is one which will necessarily be determined by many factors in any individual case. Such factors will be chiefly the inclination of the individual and the amount of thoracic work which comes to him. At the present time thoracic surgery is so new that the opportunities for strict specialization in it are very few. In time, however, along with the growth of the field and with the education of the general medical profession in the better diagnosis of thoracic diseases, and in the possibilities of relief offered by surgical attack there will unquestionably be more opportunities for specialization in this work. Experience in the past has shown that the foundations of the special fields have been built by those who were not specialists. After the foundations have been laid, the general medical profession becomes aware of the possibilities offered by the special field. Patients in great numbers are recognized who belong to that special category, and for that reason the opportunity arises for one to devote his entire time to the particular field. In thoracic surgery almost certainly the same course of events will follow. For example, when this Association was formed, in 1917, there was not one member of it who was a specialist in this field. Now there are a few, but as time goes on there will certainly be many. The question, therefore, has very naturally been raised by your President as to whether or not consideration should be given to the formation of a qualifying board in thoracic surgery. In this connection it will be appropriate to give some information about the proposed American Board of Surgery, particularly because the question of the qualification of surgeons in special fields of surgery, such as thoracic surgery, will come up before the Board.

It happens that I am chairman of a committee appointed by the American Surgical Association last year to investigate the desirability of the creation of an American Board of Surgery. This committee of the American Surgical Association met with representatives of the Surgical Section of the American Medical Association, of the American College of Surgeons, of the Western Surgical Association, and of the Southern Surgical Association. As a result of this combined meeting a plan for the creation of an American Board of Surgery has been drawn up. This plan will be presented to various national and regional surgical associations for their approval. These associations are the American Surgical Association, the Surgical Section of the American Medical Association, the American College of Surgeons, the Southern Surgical Association, the Western Surgical Association, and the New England Surgical Society. It would carry me too far afield to go into a detailed discussion of what this proposed plan is. It will be sufficient perhaps

for the purposes of the present discussion to state that the proposed plan calls for a board of thirteen members allocated as follows:

| | |
|---|---|
| The American Surgical Association | 3 |
| The Surgical Section of the A. M. A. | 3 |
| The American College of Surgeons | 3 |
| The Southern Surgical Association | 1 |
| The Western Surgical Association | 1 |
| The Pacific Coast Surgical Association | 1 |
| The New England Surgical Society | 1 |
| | 13 |

It will be the province of this proposed board to grant certificates of qualification in surgery. For all those who will not belong to the Founders Group examinations will be required covering the field of general surgery together with that much of the preclinical sciences as is necessary for the proper understanding and practice of surgery. It is the plan to reserve the last afternoon of a three-day period of examination for a more intense examination in a special field. For example, one who wishes to obtain a qualifying certificate in the specialty of thoracic surgery, neurosurgery or plastic surgery may do so by taking a special intensive examination in that field. It is proposed that, as a preliminary requirement, before a candidate can take an examination by the Board, he must have shown evidence of having had a rather sound training in general surgery. Briefly, this will consist of a minimum of four years' experience in the hospital after graduation and another period of two years of either practice or training; so he will not be allowed to appear before the Board in less than six years' time after graduation. Special emphasis in the training is placed upon the necessity of opportunities of actual operative work. I have purposely omitted the presentation of many of the details of the plan because of the limitations of time. Many of these details will also not be determined until after the Board has become organized and has drawn up its constitution and by-laws. It is uncertain, therefore, at the present time just what the detailed arrangement will be if the American Board of Surgery becomes an actuality. The principle, however, seems a sound one, and for a good many years to come the question of the qualification of specialists, like thoracic surgeons, could apparently be very well handled by such a Board.

Perhaps the American Association for Thoracic Surgery may feel that the responsibility for the qualification and certification of thoracic surgeons should rest with it rather than with the proposed American Board of Surgery. If such is the case, certain possibilities present themselves. One is that the present members of this Association who are surgeons might be certified by this Association as qualified thoracic surgeons. In the future only those would be certified who had been

qualified in general surgery by the American Board of Surgery and who, in addition, had passed a special examination conducted by this Association. The plan just mentioned would involve the creation by this Association of a special examining and qualifying board which could function as an auxiliary to the American Board of Surgery, receiving for its consideration those candidates appearing before the latter board who might express their desire to become qualified in thoracic surgery. There would probably be no difficulty in arranging an entente cordiale, such as would be implied, after the American Board of Surgery becomes organized. Another possibility would be for this Association to handle all matters of qualification and certification of thoracic surgeons entirely independently and with no regard whatever for the American Board of Surgery. It would seem to me that the former plan is to be much preferred over the latter. In the meantime it might be well for a special committee of this Association to consider what the qualifications of a thoracic surgeon should be and to recommend to the Association the ways and means for the certification to the public of those who are reasonably qualified in this special field.

# THE TRAINING OF A SURGEON WHO EXPECTS TO SPECIALIZE IN THORACIC SURGERY*

JOHN ALEXANDER, M.D.
ANN ARBOR, MICH.

MANY major thoracic operations are being performed today by surgeons whose only preparation for the work has been the reading of articles and books, and a visit of several days, weeks or months in a thoracic surgery clinic. These surgeons are learning a specialty almost solely by the trial-and-error method which necessarily yields a shockingly high proportion of deaths or other unsatisfactory results. The inevitable effect of this situation is to impress upon many internists that the surgery of the diseases of the chest is exceptionally dangerous and that the clinical results among the surviving patients are poor. Furthermore, the high mortality and the few good results tend to scare patients, especially those grouped in sanatoriums in which bad as well as good news travels rapidly, into refusing to have operations that are clearly needed. Unless this situation can be changed by the training of an adequate number of competent thoracic surgeons, thoracic surgery is likely to suffer a serious, prolonged setback which would be most unfortunate after the just popularity earned for it by the painstaking work of the pioneers.

Thoracic surgery has become so complex, and the details pertaining to the proper selection of patients and operations, and to operative technic and preoperative and postoperative management have become so many that consistently good results can be obtained only by those surgeons who have special knowledge of thoracic disease, anatomy, pathology and physiology, and a broad practical experience with the surgery of the chest. Since there are now clinics in which these things may be learned under supervision, the time has passed when surgeons must gain their experience at the expense of their patients.

The extent to which a surgeon should be trained before he begins the independent practice of thoracic surgery is subject to wide differences of opinion. It has been my experience that less than two years of intensive training in a very active thoracic surgery clinic is insufficient to give a student the necessary self-confidence later to manage the problems of his practice and to serve as a competent consultant whose opinions and surgical ability internists will respect. A greater length of time than two years would be desirable but is unnecessary. The temptation is, however, great on the part of the clinic to retain its postgraduate

*From the Department of Surgery, University of Michigan Medical School.
Read at the Nineteenth Annual Meeting of the American Association for Thoracic Surgery at Rochester, Minnesota, May 4, 5, 6, 1936.

students after the second year because they are then qualified to take a full share in the responsibility of running the clinic. Such a prolongation of the normal course of study is, I feel, unjustifiable, inasmuch as there is an acute demand for trained thoracic surgeons and because there are at present only a few thoracic surgery clinics offering a limited number of openings for training to an increasingly large number of applicants.

The Council on Medical Education and Hospitals of the American Medical Association in its pamphlet, "Essentials for Approved Examining Boards in Specialties," revised to June 9, 1935, does not recognize thoracic surgery as a suitable field for the certification of specialists in spite of the fact that there are at present a large number of surgeons specializing in thoracic surgery and that the American Association for Thoracic Surgery has been an outstandingly active organization since it was founded nineteen years ago. With regard to the twelve specialties listed, including obstetrics and gynecology, urology, and orthopedic surgery, a minimum of only a year's interneship is required before the applicant may begin his special studies. His training must include not less than three years' work in institutions approved by the Council, and "an additional period of not less than two years of study and/or practice." I am convinced that such a division of time is not suitable for the training of a thoracic surgeon. I now feel that not less than two years of active hospital work in general surgery after the interne year should be required before the beginning of special study and that two years of intensive study and practice in an active and not overstaffed thoracic surgery clinic are sufficient to qualify an applicant to take the examination of a board. It is, however, obvious that a student requiring two years in which to gain a sufficient breadth of diagnostic and surgical experience in a clinic in which an average of thirty thoracic operations are performed each week might, on the other hand, require five years in a clinic in which only ten thoracic operations are performed each week. Since the practice of thoracic surgery is frequently concerned with extraordinary situations, only broad experience with a large number of patients can prepare a student for the conditions he will be called upon to face alone in his own practice.

Since there are many more applicants for training than there are openings for them, the surgeons in charge of thoracic surgery clinics are in a position to select only highly desirable students who have graduated from class A medical schools, who have had interneships and at least two additional years of surgical work in high-grade hospitals, and preferably students who have already had some special training in internal medicine, sanatorium practice, anatomy, pathology, physiology, and laboratory methods. It is obviously desirable that those accepted for training should be in sufficiently good health to withstand the exacting responsibilities which are an integral part of thoracic

surgery, and that they should have a genuine interest in the subject apart from its offering an uncrowded field for practice. Furthermore, they should possess temperaments and personalities that are likely to contribute toward success in this specialty.

In view of the present great need for experienced thoracic surgeons I feel that the chief emphasis during the two or more years of training should be placed upon intimate contact between the student and as large a number of patients as possible, and that investigative work and study of the basic sciences, important though they are, should be given secondary consideration. Nevertheless, in the selection of students, those who are interested in investigation and who are familiar with laboratory methods should be given the preference over other equally well-qualified applicants.

Since I have been delegated to outline a course of training that I consider proper for a surgeon who expects to specialize in thoracic surgery, and since I am not familiar with the details of the training offered by thoracic surgery clinics other than Doctor Haight's and mine, I shall outline the nature of the work done by the postgraduate students in our clinic. The system of training we now use seems to us more satisfactory than any we have used in the past.

The students immediately become members of the thoracic surgery staff and perform resident type of work, although they are not literally resident in the hospital. They share in an increasing degree, as their experience increases, in the staff's responsibilities for both undergraduate and graduate teaching and in the diagnosis, preoperative and postoperative management, and the operative work on all our patients, both ward and private. This work, together with the other duties to be described, occupies an average of nine or ten hours a day during two years, except for two vacations of a month each. During the first three months the students serve as assistants in the operation room. Next they begin independently to perform the simpler thoracic operations, at first under the direct guidance of a preceptor, and during the second year they independently perform virtually every type of major thoracic operation. By the end of the two years' period of training a student will have performed from 300 to 500 operations and bronchoscopies, and will have served as first assistant in approximately twice this number. The student who has acted as the operating surgeon or first assistant is primarily responsible for the diagnosis and the preoperative and postoperative care of his patients, though under the direct supervision of the senior members of the staff, and with the aid of the assistant residents and internes.

Throughout the day the student is in virtually hourly contact with one or both of the senior members of the staff. The staff as a whole consults daily about the roentgenograms pertaining to all the patients on the service and joins in frequent consultations and conferences with

the staffs of neighboring sanatoriums and with the internists and roentgenologists of our own hospital.

In the first year of training the students make a complete dissection of the thorax and of the neighboring portions of the neck and abdomen. Microscopic slides of pathologic material removed at operation become available for study in the surgery staff room the second morning after operation. The modern concepts of thoracic physiology are studied in relation to the many physiologic problems the patients present and in individual investigative studies. Throughout the two years of training the students read widely in the literature of thoracic surgery and are expected to have a reading knowledge of medical German, and it is hoped they may also be able to read French, Italian, and Spanish. Each student is expected to do not less than one piece of original investigative work in either the laboratory or the clinic and to publish his findings. At present most of the students pursue special work in roentgenology, physiology or anatomy which, together with that in thoracic surgery, leads to the degree of Master of Science in Surgery.

It has been my experience that able general surgeons who have had such a course of special training as has just been outlined acquire sufficient knowledge and independence of judgment to practice thoracic surgery with confidence in their own opinions and in their ability to produce first-rate results, and that these men make competent consultants.

The clinical results from thoracic surgery in a great many communities in this country are inferior to what they should be simply because much of the work is at present being done of necessity by untrained men. The need for thoroughly trained men is so great that every thoracic surgery clinic of sufficient size should, in my opinion, undertake the special training of postgraduate students rather than spread all the work among the rotating residents and internes who thereby acquire too little knowledge and experience to be useful for the average thoracic surgery patient they may meet in their practices. I believe that until more of those members of this association who have active thoracic surgery clinics undertake to offer intensive courses of adequate length to as many qualified applicants as possible, and until an examining board is established to certify thoracic surgeons as fully competent specialists, due progress in the practice of thoracic surgery will be long delayed.

# THE TRAINING OF THE THORACIC SURGEON FROM THE STANDPOINT OF THE PHTHISIOLOGIST*

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THE phthisiologist is directly interested in the training of thoracic surgeons because he desires a well-equipped surgeon within call. By well equipped he means not only one who is technically proficient but one who understands tuberculosis and the problems which arise in the course of this long, chronic illness. He and his patient depend upon the judgment and skill of the good surgeon literally to save the patient from death. A poorly trained and ill-equipped thoracic surgeon may leave much wreckage and disaster behind him. The demand for a well-trained thoracic surgeon is growing daily as the advantages of surgery in tuberculosis are being better understood.

The question before us is how may a surgeon best be trained in the specialty of thoracic surgery. A review of the present situation in regard to tuberculosis, in which field the bulk of chest surgery is performed, may help to answer this question.

A recent survey† shows that in the United States there are 471 tuberculosis sanatoriums and 740 departments for tuberculosis in general hospitals, hospitals for mental cases, prisons, etc. The bed capacity is in the neighborhood of 100,000 of which 65,000 beds are in sanatoriums and 28,500 beds in general hospitals. In 1934 there were 67,000 patients with frank pulmonary tuberculosis cared for in these beds, of which 29 per cent were classified as moderately advanced and 57 per cent as far advanced; a total of 58,000 patients in the two groups in which most of the thoracic surgery is performed. Reports from 500 of the 1,240 institutions show that in 1934 there were approximately 3,000 thoracoplasties, 1,000 pneumolyses, and 7,500 phrenic nerve operations performed.

The surgeons who perform these operations may be divided into three groups:

1. The full time thoracic surgeon. He is located in the large center or in a place that draws from a wide area. He performs all types of surgery on the chest and its contents, including tuberculosis. He has had a thorough training in general surgery but now devotes his entire time to the thorax. Those who plan to devote their full time to surgery of the chest must be well grounded in tuberculosis. This means long

*Read at the Nineteenth Annual Meeting of the American Association for Thoracic Surgery at Rochester, Minnesota, May 4, 5, 6, 1936.

†Tuberculosis Survey, J. A. M. A. 105: 1855, 1935.

training, and probably only the younger men not yet fully established in practice can undertake such a course as outlined by Dr. Alexander.

2. The second group is composed of general surgeons who are interested in the thoracic field. Probably the bulk of the surgery in tuberculosis is performed by this group of men. Many are our most proficient lung surgeons, but in many instances this is not the case, and it is the latter type of surgeon who needs further training before he performs these major operations on the thorax. But how to train him is the problem. We can visualize a surgeon well established in practice, standing high in his profession, who is asked by the head of a neighboring tuberculosis sanatorium to undertake the chest surgery of that institution. Without proper knowledge of the situation he may be inveigled into performing a thoracoplasty with possible disastrous results not only to the patient but to the cause of lung surgery in the community. Such a surgeon cannot leave his practice for a two to four year period of training, but he can gain an increasing knowledge of tuberculosis by attaching himself to the staff of the neighboring sanatorium. He should take part in the discussion of all cases and by following the individual patients learn the vicissitudes of the disease. If the medical staff is surgically minded, he becomes acquainted with their opinions in regard to the part which surgery plays in the attempt to stop the progress of the disease. The surgeon should know the physiology of the lungs and the thoracic cage. He should know the different types of cavity formation and their probable behavior, acquaint himself with the resistance or lack of resistance shown by the patient, be able to interpret roentgenograms of the lungs, and be familiar with the numerous other factors which are daily met with in tuberculosis. He should attend the large surgical clinics in chest diseases and study the technic of the various operators, and if possible assist at such operations. This program represents the very minimum in requirements, but the well-endowed general surgeon can acquire fairly quickly the principles of thoracic surgery.

Lack of teamwork between the internist and the surgeon is to be deplored. For a surgeon to undertake a thoracoplasty upon the say-so of the internist is bad surgery, and for the surgeon to tell the internist what patients should be operated upon is equally bad. Unfortunately there are many physicians in charge of thousands of tuberculosis patients who are not yet awake to the benefits of thoracic surgery.

3. The third group of thoracic surgeons is a small one. It is composed of those heads of institutions who also perform their own surgery. Even if the sanatorium is a large one, there is not sufficient surgery of the chest to be performed so that the operator can keep technically proficient. I know that in some instances the work is well done, but medical men should not undertake thoracic surgery as a side line.

Many young men are in training, and in a few years they will be the thoracic surgeons of the country. But phthisiologists will still have to

depend to a large extent upon the general surgeon who is interested in thoracic surgery to perform their operations. Possibly this association will establish minimal requirements which would act as a guide for these general surgeons who desire to undertake thoracic surgery on the tuberculous.

There is one point which bears upon the subject under discussion and that is whether tuberculous patients should be referred to general hospitals or whether the surgeon should perform the operations at the sanatorium. Relatively few institutions in the country are properly equipped and have an adequate nursing staff to take care of patients undergoing these major operations on the chest. The after care is so important that the operating surgeon should be within close call or else the staff of the sanatorium should be so well trained that they can take care of the emergencies as they arise. Outside of the few very large centers where all of the surgery is done at the sanatorium, the majority of patients are sent from the sanatorium to the general hospital for their surgery. Because of the cost of maintaining operating room facilities and a surgical staff, this method of caring for the surgery in tuberculosis will probably continue for some years to come.

The time has arrived when every sanatorium should have a first class thoracic surgeon on its staff, which means we must have more and better trained surgeons in the field of tuberculosis.

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## DISCUSSION OF SYMPOSIUM ON THE TRAINING OF THE THORACIC SURGEON: "SPECIAL TRAINING FOR THORACIC SURGERY," BY EGGERS; "FROM THE STANDPOINT OF THE GENERAL SURGEON," BY GRAHAM; "FROM THE STANDPOINT OF THE THORACIC SURGEON," BY ALEXANDER; "FROM THE STANDPOINT OF THE PHTHISIOLOGIST," BY PACKARD

DR. LOUIS B. WILSON.—This subject is one which concerns us so much here in the Mayo Foundation that I am constrained to invite you to go one floor up in this building, at your convenience, and see the general plan of graduate education in the various fields of medicine which the Mayo Foundation has been carrying on through the University of Minnesota for the last twenty-one years. Many of the things which have been proposed here today are directly in line with the work which we have been doing, and it is possible that our experience might be of some suggestive interest to you. If you will come either as individuals or as groups, we shall be very glad to welcome you and will attempt to answer any questions you may ask.

I should like to go further, just for a moment, concerning the standards which were set up by the Council on Education of the American Medical Association and the Advisory Board of the Medical Specialties and finally approved by the House of Delegates of the American Medical Association. There was no intention that these should be other than the bare minimum requirements. Further, the twelve specialties which were set up by the Committee on Standards of the Advisory Board were intended in medicine and in surgery to include subdivisions, which it was assumed might follow some set-up such as Dr. Graham has suggested in his plan No. 1.

I may suggest to the surgical group who are concerned with the possible formation of a board in surgery that the Board of Internal Medicine has already recognized that subdivision in that field is necessary, and has stated in its first announcement that they are concerned first with the certifying of men in internal medicine, but that they recognize that there must be certification, probably after July 1, 1937, in subdivisions of internal medicine, such as gastroenterology, cardiology, and so forth, but that this should be done only by some form of subsidiary or accessory boards composed of men who are familiar with these particular fields.

It all comes back to the basic principle enunciated by the original committee studying this problem, that only men who are competent in a specialty are competent to say who is competent in that specialty. Now, it is perfectly possible for any board, as a board of thoracic surgery, to accept first of all the requirements, all or a portion of them, of the board of general surgery. When once the board of surgery is established, then the board of thoracic surgery may be established.

May I go one step further, and call attention to one thing which it seems to me has not been sufficiently emphasized. That is the importance of a provision for a much longer training service than is now possible in most institutions. Dr. Alexander said it was doubtful whether brief services in this or that special field were desirable. It seems to me that they are most undesirable. The Committee on Standards of the Advisory Board has definitely in mind to ask teaching institutions that have control of good hospital facilities to reduce the number of residencies, but increase the length of their services. That was the first thing which was done toward graduate work in this institution. Thirty years ago we changed the internships from one year to three years, and soon after required a year of interneship before residencies. There are very few institutions that have adequate provision for longer residencies, in general surgery, in general medicine, or in any subdivisions of medicine, and today it seems to me that in your home institutions that is the most difficult problem you have to meet and one which is most needing to be solved.

DR. HOWARD LILIENTHAL.—Perhaps I should not speak on this subject, because my own education in surgery has been quite different from that of the present time. History, however, may be worth while; and I may point out one or two aspects which may not have struck the younger men.

If we wait too long before we are permitted to practice thoracic surgery, or any other branch of surgery for that matter, we shall find that with the retiring age from hospital service of about sixty-two, there will be disproportionately few years in which to practice. Undoubtedly we shall enjoy our period of education in the special work, with Dr. Alexander, with Dr. Graham, or with the Mayo Clinic, to mention but three examples. It appears to me, however, that honesty of intention is the most important factor in determining who should practice. I recognize that permission for operative or other therapy is granted by our present laws, and that the alterations which are here proposed would not be compulsory unless changes were made in our laws. I maintain that there is no reason why one should not enter thoracic surgery after ordinary preparation without waiting too many years for what one might call complete maturity. But the carrying out of this plan, which is the present one, should imply that form of honesty which would impel one to call a man of experience as consultant or even assistant in unfamiliar cases. This is the manner in which I myself began the practice of surgery, and I still believe it to be morally efficient. These remarks are not intended to belittle the advantage of education in clinics and hospitals, but work of this kind, preliminary to practice, should not be too long.

Thoracic disease may have to be approached through the abdomen, another reason for the importance of general surgical preparation. Dr. Harrington showed me today some amazing and beautiful motion pictures in which both cavities were

invaded. Thoracic surgery does not consist merely of rib cutting, as some seem to think. One who wishes to enter the field should be what I call *thorax-minded* —an expression similar to the term "air-minded," which has been coined by the Army. It describes a condition indispensable if one is to become a pilot. A fine pilot of an airplane will be examined most carefully, psychologically and otherwise, and if he is not found to be air-minded, he is not accepted as an Army aviator. There should be some method outside the usual forms of examination to reveal when a surgeon is thorax-minded.

A capable general surgeon may be called upon in acute cases to act in an emergency. A stab wound of the heart must be repaired by the first surgeon at hand, though he belong to any specialty—gynecology, even ophthalmology. The patient must not die because of the absence of conscientious effort.

Before closing, let me bring to your attention once more the founder of this Society, the lamented Willy Meyer. His great wisdom was demonstrated when he founded the Association for Thoracic Surgery and included in its membership other branches of medicine—roentgenology, pathology, endoscopy, and general medicine. It was he who pointed the proper way for the training of the thoracic surgeon.

DR. ETHAN FLAGG BUTLER.—It was a happy thought on the part of our president to bring up the present discussion. At the present moment I am responsible for inaugurating a surgical program in three new tuberculosis hospitals in New York State. Speaking personally and not officially, it is my great desire to see men who are thoroughly grounded in the principles of general surgery installed in the responsible surgical positions in these hospitals. I shall go home from this meeting strengthened in my belief that the thoracic surgeon must have a very thorough training in general surgery.

There is an economic aspect to the practice of thoracic surgery that cannot be ignored. It is fair at this time to warn the young man who hopes to make thoracic surgery his one and only field of professional endeavor that 75 to 90 per cent of all thoracic surgery must be done without hope or expectation of financial remuneration. Those, therefore, who engage in this work to the exclusion of all else must either be subsidized or independently wealthy. Before a young man commits himself exclusively to the field of thoracic surgery, he should ask his older friends what it has cost them to maintain their interest in this field.

DR. POL N. CORYLLOS.—I shall present a few opinions which are drawn from my own opportunities in this country, about the way in which we should try to supply the acute demand for trained men.

In 1931 I was asked to direct the organization of a large sanatorium near New York City, with 1,700 beds, with another service of 500 beds which represents a division of a hospital in New York City. In both institutions about six thoracoplasties had been done in the five preceding years. There was no other organization to take care of that surgical treatment in that city. The question came to me how I was going to organize these services and what were my duties toward the formation of the staff with men who would be able to take adequate responsibilities as cooperators for that work.

I came to the conclusion that the best way to handle the question would be to take as residents only men who had at least three years' surgical instruction, so they would be thoroughly grounded in general surgery. It has not been difficult to find these men. Second, to train them for six months in physiology, in anatomy of the chest, and to some extent in experimental work, and after the six months to allow them to go ahead with minor operations. As to minor operations, unfortunately there are very few of them, but I mean second stage or third stage thoracoplasty, empyema operations, and so forth. After the first year was over, to allow them to go ahead and operate under my supervision, to start with major operations, and at the end of six months more to do all surgical work.

The question was whether I should give them work to do alone, so that when they get out they would have the courage to operate upon the unsuspecting public, or give them enough work so that when they go out they will be able to take full responsibility. I arrived at a decision that every one of my residents should get out with a minimum of forty thoracoplasties done by himself. I think we have here in this room one of my most brilliant residents, who had done ninety thoracoplasties when he went out. I think that if we all take that position, the problem will be solved, and you are going to be able to furnish men with an adequate surgical background and with thorough training in thoracic surgery. I suppose that the minimum standards required will satisfy the requirements of an adequate training; by doing that a sufficient number of operations will be provided to cover at least part of the necessities which are presented because of the tremendous expansion of this field, and take away the difficult operations from men who do not know how to do them, because do not forget that there are among our profession men who today will do anything. We are all seeing disasters with simple operations.

Those are a few opinions which are very harmless to give to the society, and I think the men here represent certainly fully 50 per cent of the big institutions in the country. Perhaps if we do this, we are going to give to the country a sufficient number of surgeons to cover the necessities of the present.

DR. W. A. HUDSON.—This is a question that has been of some interest to me, because I happen to have been one of the first to break away from general surgery and limit myself to thoracic surgery. I know some of the difficulties that a man who limits his work to one line has to overcome. I realized from the beginning that thoracic surgery was developing into a rather broad field and that the limitations of the thoracic surgeon depended in a great measure not upon the field itself but upon the man who entered it, and that if he intended to make a living in the practice of thoracic surgery he should be capable, through experience gained by the cut-and-try method, or by going here and there and picking up whatever bits of information he could, to meet any problem that might arise within his practice.

I have found that there is no difficulty in getting plenty of work to do. In fact, once one establishes himself in a community in which there is a large population, one will find that he will have all the work that he can do and will have no slack time on his hands. Dr. Butler brought out one point that is a rather important one. I think, however, that he set his estimate rather low when he said that 90 per cent of the work would be without remuneration. I am certain, Dr. Butler, that you must have crowded in a little general surgery. I think, however, that in part the lack of remuneration is our own fault. I believe as the general practitioners become better acquainted with the possible help to be had from the thoracic surgeon and the need of calling early for help, that the class of patients who are now unable to pay their way will be able to pay the thoracic surgeons something for taking care of them, in the same manner as they now pay the general surgeon.

There is, however, another side to this subject that I am more interested in than in this question of whether the surgeon is paid for his work. After all, the patient should come first if we are to take care of him, and I believe that the man who does not have a good foundation on which to start will not be capable of developing into a good surgeon, whether it is a general surgeon or a thoracic surgeon. I would entirely agree with Dr. Butler in his statement that he would rather have a man who has had a good foundation in the fundamentals of surgery and the general sciences, without any special training, than to have a man who has neglected to obtain those fundamental and basic principles but has had a lot of experience in observing and even assisting at special operations.

I think, Dr. Coryllos, that I would not go so far as to set up a minimum or a maximum number of operations that my assistant should assist at or do himself. I would be inclined to size up that assistant from a standpoint of his personality and his capacity to absorb and profit by whatever came his way. I think that is the angle from which we shall have to estimate that individual's ability to undertake these specialties.

DR. HARRY C. BALLON.—I should like to say a few words about this subject, not because I spent many years in obtaining a training before I engaged in private practice and therefore feel that a lengthy period of training is necessary, but rather because it appears to me that much of the discussion has missed its mark. I do not regard whether a man will be able to make a living from the practice of thoracic surgery as the prime consideration in the discussion; certainly not at the present time, since it has not been suggested that he limit himself to the practice of this particular specialty.

I believe it is safe to say that we are all in entire accord with the statement that no one should undertake to practice thoracic surgery, as we know it today, without having had some special training. Obviously then the all-important point is, what constitutes an adequate training. If a decision is reached about this point, about the minimum requirements, then one can consider whether or not a sufficient number of clinics provide opportunities for obtaining such a training. If facilities are few and applicants many, then the former must either be increased, or the minimum time period requirements reduced. A move in the latter direction would probably be unwise and might defeat the purpose. It is appreciated that some can acquire information more rapidly than others. Neither Beethoven nor Bach were bachelors of music yet even they required a training in music.

I repeat then that the most important consideration under discussion is, what constitutes an adequate training. There are untrained surgeons who do the same operation many times, each time poorly and yet call it experience; others, well trained, do the same operation on fewer occasions, yet know how to do it.

To consider the standards of twenty years ago and to say that what was adequate in the way of training then suffices at the present time would be unfair, since thoracic surgery has progressed so much during the past two decades. It is to my mind not only desirable but essential that a training in any particular surgical specialty be preceded by a training in general surgery.

If it comes to be known that the surgeon who engages in the practice of thoracic surgery has had a recognized and approved training in this specialty, then opportunities should be more numerous and more satisfying than they are at the present time.

# WOUNDS OF THE HEART*

## Report of Thirteen Cases

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THE suture of wounds of the heart is a modern surgical attainment. Delay in its successful accomplishment was no doubt due to the pronouncements of the medical masters of the past, all of whom believed that heart wounds were necessarily fatal and that intervention was useless. Homeric literature abounds with references to fatal cardiac wounds; Hippocrates[1] believed them to be deadly. Paré[2] described them but made no suggestions regarding their treatment. Boerhaave[3] stated that all wounds of the heart were mortal. Hunter made no mention of heart wounds, and John Bell[4] in his *Discourses on the Nature and Cure of Wounds* devoted only two paragraphs to the subject, although he recognized the signs and symptoms of the condition and cited two patients who lived several hours and might well have been saved by operation. He thus dismissed the subject: "there is so little to be done . . . and the signs and consequences are so clear, that it were a waste of time to speak longer of wounds of the heart." Billroth,[5] who dominated the surgery of his day, wrote (in 1875): "Paracentesis of the pericardium is an operation which in my opinion approaches very closely to that kind of intervention which some surgeons would term a prostitution of the surgical art and others madness." He did add, prophetically, "Perhaps another generation will think otherwise about it." Later (in 1883) he made the statement that "the surgeon who should attempt to suture a wound of the heart would lose the respect of his colleagues," and as late as 1896 Stephen Paget[6] wrote: "The surgery of the heart has probably reached the limits set by nature to all surgery; no new method and no new discovery can overcome the natural difficulties that attend a wound of the heart."

Two figures stand out because of their disagreement with these pessimistic pronouncements; Morgani,[7] who in 1761 showed that blood in the pericardium compressed the heart and embarrassed its movements, and Baron Larrey[8] (Napoleon's great surgeon), who decompressed a wounded heart by drainage, and who demonstrated by experiments on dogs that these injuries were not necessarily fatal.

Modern surgery of the central circulatory system began with Roberts[9] (1881) who suggested that wounds of the heart be sutured. Block[10]

*From the Department of Surgery, Emory University.

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(1882) successfully sutured the hearts of rabbits, but Del Vecchio[11] (1895) deserves the greater credit for demonstrating to the Eleventh International Medical Congress at Rome the healed wound in a dog's heart. Within a year the human heart was sutured by Cappelen[12] (September, 1895), by Farina[13] (March, 1896), and by Rehn[14] (September, 1896), the last case surviving. By 1909 Peck[15] was able to collect 161 cases treated surgically, with a mortality of 63 per cent. Pool[16] (1912)

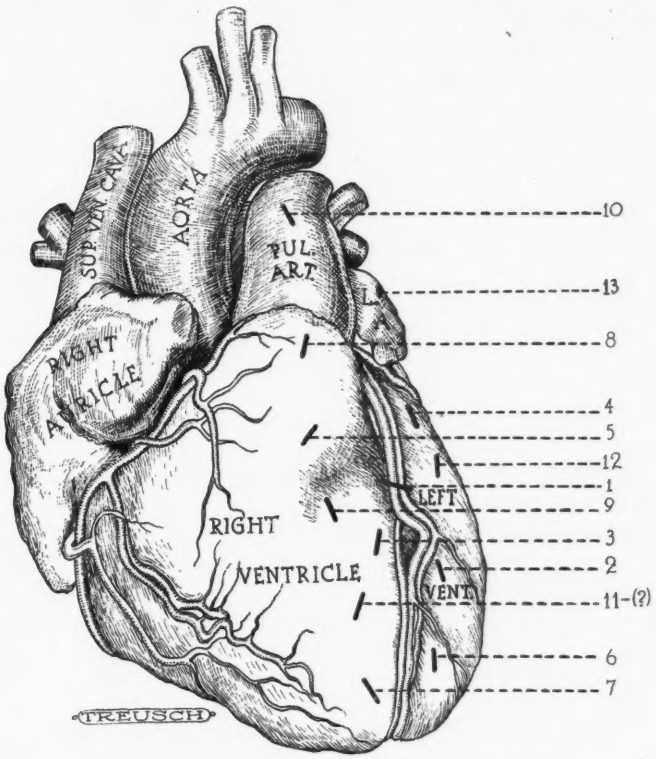


Fig. 1.—Illustrating the approximate point of cardiac injury in the 13 cases described in the text. The numbers correspond to the case numbers.

added 79 cases, with a mortality of 49 per cent; and Smith[17] (1923) added 58, with a mortality of 33 per cent. Schoenfeld[18] (1926) collected 25 cases, with a mortality of 36 per cent; and Bigger[19] (1932-1933) added 70 instances with a mortality of approximately 30 per cent.

The percentage of reported recoveries is unquestionably too high, because of the fact that many single favorable cases have been reported, whereas those ending fatally are not so likely to be recorded. At any rate, the percentage of recoveries is at the present time at least 50 per

cent, whereas only 10 per cent of the untreated cases reported by Fischer[20] (1868) recovered.

The purpose of this paper is to report thirteen instances of cardiac wounds which I have personally treated, and to discuss certain features of diagnosis and treatment.

*Diagnosis.*—Wounds of the heart are relatively rare. At the Medical College of Virginia Hospitals[19] 0.1 per cent of surgical patients had such injuries, and, in my own experience, approximately 2 per cent of penetrating wounds of the chest injured the heart. Considering its size and exposed position, it is surprising that it is not more frequently injured. If those dying of suicide or homicide and never reaching the operating room were considered, the percentage would probably be much higher.

Every attempt at accurate diagnosis should be made before operation, since exposure of the heart is in itself hazardous. However, if it is impossible to determine whether or not such an injury exists, it is safer to do an exploratory pericardiotomy than run the chance of death from heart tamponade or hemorrhage, or both. All the patients in this series had tamponade of the heart, and in the majority there was a history of hemorrhage to the outside or blood was found in large quantities in the pleural cavity. It is likely that some patients die of severe hemorrhage without the establishment of a tamponade, but I have not encountered this condition at operation.

The position and the direction of the external wound are of some importance in diagnosis, but the course of a bullet or a knife thrust is notoriously misleading, although those just to the left of the sternum from the second to the fifth rib are most likely to injure the heart.

The history is of importance, for it is usually characteristic. Bleeding is profuse at first, with no other symptoms for several minutes. During this period the patient feels no evidence of injury. One of my patients ran three blocks (Case 1); another walked two blocks (Case 3); and a third was able to fight for ten minutes (Case 7). This is followed by exhaustion and collapse, frequently with unconsciousness. At the same time there is usually a cessation of bleeding from the external wound (Cases 3-11). This chain of symptoms is due to a gradually developing tamponade. When the heart is wounded, it bleeds freely to the outside or into the chest. At the same time blood collects in the pericardium and some of it clots. When 100 or 200 c.c. has collected, the pericardium becomes distended, the intrapericardial pressure rises, and the venae cavae can no longer empty blood into the heart. The heart, being unable to fill to full capacity, can no longer empty, and cerebral anoxemia results. Contractions are feeble, sometimes irregular, and usually slowed. The latter is probably the result of reflex vagus action.

The patient gradually becomes irrational or comatose. The skin is cold and moist. Heart sounds are indistinct, and the pulse is weak or

absent. The arterial pressure is lowered or imperceptible, and the venous pressure is raised as evidenced by prominent, strutted external jugular veins. Because of venous stasis, there is a marked pallid cyanosis of the lips and tongue.

Roentgenogram of the heart is of no value in diagnosis, since death may occur from a rapidly developing tamponade from an amount of blood too small to cause a noticeable change in the size and contour of the cardiac shadow.

Fluoroscopic examination, as pointed out by Bigger,[19] is of great value, since an accumulation of blood in the pericardial sac prevents the normal pulsations of the heart. Three patients recently seen and suspected of cardiac injury were not operated upon following fluoroscopic examination and all recovered. Electrocardiograms are of little value, since normal tracings may be obtained for several hours after the injury.[21] Aspiration may be used where diagnosis is otherwise impossible, or on patients in extremis, in order to decompress the heart temporarily.

After a diagnosis is established, immediate operation should be carried out. The dangers of infection and the frequent deaths from this cause demand the most meticulous care in preparation, and in carrying through the operation under a rigid aseptic technic. While preparations are being made for the operation, the patient should be kept as quiet as possible by the administration of morphine. External heat should be applied, and the patient's head should be lowered. Saline infusion, transfusion, and measures which tend to raise the blood pressure are of no value in cases of tamponade and may be of harm in increasing hemorrhage, but intravenous administration of a 6 per cent solution of acacia should be given when the operation is begun. Autotransfusion of citrated blood from the chest and pericardium should be given during and after the operation if there is little danger of the blood being infected, and a blood transfusion should be given as soon after operation as is possible.

Since the pleura is usually injured and a transpleural approach is frequently necessary to reach the heart, nitrous oxide and oxygen anesthesia under slight pressure is preferable to local infiltration. Should local anesthesia be used at the start of the operation, inhalation anesthesia will usually be required when the tamponade is released, for with recovery of consciousness, the patient will become excited, struggle, and interfere with the procedure.

The incision should be planned to secure the best exposure in the quickest time and with the least shock. The median sternotomy (Duval-Barasty) gives excellent exposure to all the heart and the great vessels, but splitting the sternum requires a great deal of time, as does the closure of the wound, and is productive of shock. It is mentioned only

to condemn it, since in cases of severe hemorrhage or increasing tamponade the patient would not likely survive such a procedure.

The intercostochondral thoracotomy (Spangaro) offers a rapid approach to the heart but not a particularly good exposure. It can be enlarged by cutting or removing the cartilages above and below the incision and by removing a portion of the sternum.

A third method, and one giving excellent exposure, consists of turning a flap of skin and muscle laterally and removing two or more costal cartilages. The internal mammary vessels are ligated and divided, the triangularis sterni muscle is divided and the lung and pleura displaced outward by gauze dissection. The wound in the pericardium is then located and enlarged, or, if the wound is not readily found, the peri-

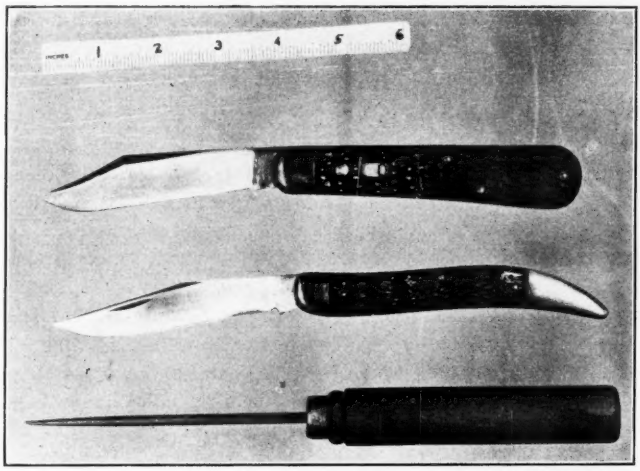


Fig. 2.—Type of weapons with which cardiac wounds are usually inflicted. In eleven of the cases described in the text the "switch blade" knife was used and in two the ice pick.

cardium is opened between stay sutures. If tamponade exists the pericardial wound is usually bleeding slightly, if at all; but, when the intrapericardial pressure is released, the bleeding becomes marked, as the contractions of the heart increase in force.

The greatest difficulty in suture is in locating the wound and in placing the first stitch. This may be facilitated by removing the blood by suction, and when the wound is located, by placing the index finger of the left hand over it. By this means the flow of blood will be impeded sufficiently to allow the passage of a suture directly under the finger. This is left untied and is held in the left hand for traction and hemostasis, and other sutures can then be readily placed to close the wound completely. They should pass well into the substance of the muscle but not into the chamber of the heart. Fine black silk on small full

curved needles is the material of choice. Under no circumstances should the finger be placed in the wound, since heart muscle is extremely friable and the wound will be torn and enlarged.

Should the wound be in an auricle or behind the sternum, or on the posterior surface, a stay suture, passed through the apex, as advocated by Ballance[22] and by Beck[23] is of great value. By this means the heart may be easily rotated.

After control of the hemorrhage, the pericardium should be carefully cleansed by flushing with salt solution, but with as little handling as is possible. The pericardium, muscle, and skin are then closed without drainage.

Immediate prognosis depends largely on the interval between the injury and the operation. Delay in operating may cause death from hemorrhage, or tamponade, or both. It likewise depends upon the character and extent of the injury; a bullet usually causes two wounds with greater hemorrhage and tissue loss.

Postoperative prognosis is dependent upon infection; purulent pericarditis and pneumonia. Six of the thirteen patients in this series died. One (Case 4) did not survive the operation and died of hemorrhage. Two patients (Cases 6 and 10) died of pneumonia on the third postoperative day. One (Case 3) died of pericarditis on the third day, and one (Case 8) lived for fourteen days and died of mediastinal emphysema. One (Case 12) died of septicemia.

CASE 1 (previously reported).—J. H., No. 43226, a negro boy of eighteen, was admitted to the hospital at 10:30 P.M., Jan. 8, 1931. A half hour previously he had been stabbed with a penknife. He ran about three blocks, collapsed, and was brought immediately to the hospital. His clothes were covered with blood, and he was bleeding profusely from a wound in the fourth interspace two inches to the left of the sternal border. He was conscious but extremely restless and begging for water. Both pupils were dilated, the extremities were cold and clammy, and the radial pulse was imperceptible. The heart sounds were weak, but could be counted, 90 per minute. The rate soon dropped to sixty and the external bleeding stopped. The blood pressure could not be determined. Electrocardiogram, made before operation, showed no characteristic deviation from normal.

A diagnosis of cardiac tamponade from stab wound of the heart was made and immediate operation performed. Under procaine anesthesia a flap of skin and muscle from the second to the fifth left costal cartilage was turned outward. The third, fourth, and fifth cartilages were removed, the internal mammary vessels tied, and the pericardium was exposed by pushing back the overlying pleura and lung. A wound in the pericardium, which bled with each systole of the heart, was enlarged. The contractions of the heart were weak but regular. A finger was passed behind the heart and a large amount of fluid blood and clot evacuated. Following this, the heart began to beat strongly, and the pulse became perceptible at the wrists. The heart wound, which was in the right ventricle just medial to the descending branch of the left coronary artery, began bleeding profusely. This was controlled by placing the left index finger on the wound and passing a suture of silk under the finger through the wound. This suture was then used for both hemostasis and steadying the heart while the wound was closed with two figure-of-eight sutures of silk. After closure of the wound the descending branch of the left coronary

artery and vein were found to be completely severed, and the proximal and distal ends were ligated with silk. The pericardium was closed with interrupted sutures of silk, and the skin and muscle flap replaced and sutured with silkworm gut. A small drain of soft rubber was carried down to the pericardium. The pulse at the end of the operation was 90, and the systolic blood pressure 90, diastolic 60.

The patient's temperature ranged between 99° F. and 101° F. for eight days after operation, and the pulse between 100 and 115. On the second day the systolic blood pressure was 110 and ranged between this and 120 during his stay in the hospital.

On the third day there was a definite to-and-fro pericardial friction rub which persisted until the sixth day. There was considerable drainage and on removal of the drain on the fourth day a large amount of fluid followed. A teleroentgenogram made on the fourth day showed evidence of pericardial effusion and pneumonia of the left lower lung. The teleroentgenogram made twenty days after operation was normal. The patient was discharged from the hospital on the twenty-sixth day after operation. At present (May, 1936) he is symptom free and apparently completely recovered.

CASE 2 (previously reported).—T. M., No. 41739, a negro man of twenty-five, was admitted to the hospital, Sept. 25, 1930. He had been stabbed in the chest with a knife about forty minutes previously. He was bleeding freely from a wound in the left fifth interspace near the sternal margin. He was rational, but weak and thirsty. The arterial blood pressure was 80, pulse 110, regular but weak.

Under procaine anesthesia the skin and muscles over the fourth costal cartilage were incised and about three inches of this cartilage removed. The pleura and lung were retracted laterally, and the pericardium was exposed. A wound in the pericardium, about an inch long, was bleeding profusely. This was enlarged and a wound 1.5 cm. long was discovered in the left ventricle just to the left of the descending branch of the left coronary artery. It was bleeding with each systole of the heart but was easily closed with two figure-of-eight sutures of silk. The clot and the free blood in the pericardium were then removed, and the cardiac contractions immediately became strong and regular. The pericardial opening was closed with a continuous suture of silk, and the muscle and skin layers were closed without drainage with catgut and silk.

The patient left the operating table in good condition. His pulse was 110, systolic blood pressure 90, and respirations were 30. An hour later he was given a transfusion of 500 c.c. of blood. His temperature ranged between 99° F. and 102° F. for five days after operation. Four days after the operation there was evidence of cardiac enlargement and of pneumonia in the left lower lung, confirmed by a roentgenogram of the chest. The heart sounds were diminished but regular in rhythm, the pulse was 100, and the blood pressure 115/75. The cardiac enlargement was characteristic of pericardial effusion, but since there was no embarrassment of the circulation the fluid was not withdrawn. On Oct. 15, 1930 (twenty days after the injury), there was still evidence of pericardial effusion both by physical signs and by roentgenogram, but there had been a definite decrease in the size of the cardiac shadow. The chest wound healed per primum, and the patient was discharged from the hospital twenty-six days after operation. Forty-five days after operation the teleroentgenogram was normal, and at the present time (May, 1936) he is in good condition without any evidence of cardiac damage.

CASE 3.—I. M., No. 63284, a negro male of forty-one years, was admitted to the hospital Feb. 18, 1934, at 11 P.M., with a wound 1 cm. in length in the left intercostal space 2 cm. from the sternal border. The injury had occurred about thirty-minutes before admission. He was unconscious and in profound shock. Pulse was imperceptible, and the heart sounds were weak, slow, and irregular. His lips were

pale but cyanotic. The neck veins were greatly distended. His clothes were soaked with blood, but the wound was not bleeding. There was evidence of fluid in the left pleural cavity.

Under positive nitrous oxide and oxygen anesthesia the left fifth costal cartilage and a portion of the fifth rib were removed. The pleural cavity had been entered and contained blood. The pericardium was tense and blue. A wound, 1 cm. in length, in the outer lower pericardium, was bleeding slightly. This was enlarged, and the heart was beating weakly and irregularly. About 200 c.c. of fluid blood and clots were removed from the pericardial cavity, and contractions of the heart became vigorous. A wound 1 cm. in length was found in the right ventricle, midway between base and apex, which bled with each systole. It was easily closed with three silk sutures. The pericardium was loosely closed with silk, and the muscles and skin were closed in layers.

At the end of the operation the patient was conscious, and his blood pressure was 52/0. Transfusion of 750 c.c. of blood was given, and after twenty-four hours the blood pressure was 106/70. The pulse was 90, and he was rational. He then related that after being stabbed he had bled profusely, but walked two blocks before collapsing.

Two days later he became restless and irrational, and his temperature rose gradually to 104° F. There was no evidence of fluid in the pericardium, but a pericardial friction developed.

The following day he died. Necropsy showed extensive pericarditis. There had been no bleeding, and death was due to infection.

CASE 4.—L. W., No. 68701, a negro woman of twenty-five, was admitted to the hospital Dec. 1, 1934, at 11 P.M. No history could be obtained other than that she was stabbed with a knife, bled profusely, and was brought immediately to the hospital. On entrance she was in extreme shock; the pulse was imperceptible, and the blood pressure could not be determined. The heart sounds were irregular and weak. There was a wound in the second left interspace, 2 cm. long and 4 cm. from the sternal border. This was sucking but not bleeding. There was evidence of fluid (blood) and air in the left pleural cavity. The neck veins were markedly distended, and because of this an immediate operation was performed.

An elliptical incision was made over the left anterior chest wall, and the skin and muscles were dissected laterally. The second, third, and fourth costal cartilages were removed. There was an opening in the pleura, with complete collapse of the left lung. Nitrous oxide and oxygen anesthesia was administered under pressure and the lung inflated, showing a wound through the lung overlapping the pericardium. This was bleeding and was sutured; 1,500 c.c. of blood and clots were removed from the pleural cavity.

The pericardium was tense and pulsating feebly. There was a laceration, 2 cm. long, on the left upper surface, bleeding slightly. This was enlarged, and a laceration 2 cm. in length was discovered in the left ventricle near the base. Because of the weak action of the heart, the blood and clots in the pericardium were aspirated before attempting to close the wound. When this was done, the contractions became forcible and bleeding from the wound was extensive. Five silk sutures closed the wound in the heart. The pericardium and chest wall were closed in layers. During the operation the patient was given 1,500 c.c. of saline, 500 c.c. of 6 per cent acacia solution, and 500 c.c. of whole blood. The loss of blood before and during operation was fatal, and respirations ceased as the operation was completed. With artificial respiration, the heartbeat continued for ten minutes. Death was thought to be due to respiratory paralysis from cerebral anoxemia.

CASE 5.—H. S., No. 68733, a negro male of thirty years, was admitted Dec. 3, 1934, at 11 P.M. No history could be obtained, other than that he walked into the

clinic and collapsed. His clothes were soaked with blood, and there was a stab wound 1 cm. long in the left fourth interspace 6 cm. from the sternal border. There was a pneumohemothorax on the left side. The wound was not bleeding. The veins of the neck and upper extremities were markedly dilated. His mucous membranes were cyanotic. The pulse was weak, and the blood pressure was 60/40. He was given 500 c.c. of 6 per cent solution of acacia intravenously, and immediate operation was performed under nitrous oxide and oxygen anesthesia under pressure. An elliptical incision was made on the left side extending from the second to the sixth rib. A flap of skin and muscle was reflected laterally, and the second, third, and fourth cartilages were removed. There was an opening in the pleura, and the lung was collapsed. The left pleural cavity contained about 1,000 c.c. of blood. There was an opening in the anterior lateral surface of the pericardium which was enlarged. The pericardium contained about 200 c.c. of unclotted blood. When the pericardium was opened, a wound in the right ventricle just medial to the left coronary artery was found, which was gushing with each contraction of the heart. A suture passed through the wound controlled the bleeding, and the wound was then closed with four interrupted sutures of silk. The pericardium, muscle, and skin were closed in layers without drainage, after aspirating the blood from the pericardial and pleural cavities. At the end of the operation the pulse was regular and of good quality, and the blood pressure 100/60. Following operation the patient developed an effusion in the left chest, and 500 c.c. of bloody fluid was removed on two occasions. There was a slight elevation of temperature for two weeks, but convalescence was otherwise normal, and he was discharged from the hospital forty-two days after operation. Electrocardiograms made the day after the injury showed evidence of myocardial infarction, but at the time of dismissal were normal. At the present time he is working as a laborer, without symptoms of cardiac damage.

CASE 6.—H. L., No. 69171, a negro male of thirty years, was admitted to the hospital on Dec. 30, 1934, at 2 P.M. He was unconscious, and no history could be obtained other than that he had been stabbed in the chest shortly before admission. There was a sucking wound 3 cm. long in the fourth interspace, 3 cm. from the sternum. The wound was not bleeding. There was a massive pneumothorax on the left. The pulse was imperceptible, and the blood pressure could not be obtained. The heart sounds were indistinct. The neck veins were swollen, and the mucous membranes were cyanotic.

The wound in the chest wall was sutured and operation immediately carried out under nitrous oxide and oxygen anesthesia. Through a T-shaped incision the fourth and fifth left costal cartilages were removed, and a wound in the left anterior margin of the pericardium was enlarged. A wound in the lower part of the left ventricle just lateral to the coronary artery was found and sutured with silk. This wound was bleeding very slightly and had probably not entered the ventricular cavity. One hundred and twenty c.c. of clots and fluid blood were removed from the pericardial cavity, and the contractions of the heart immediately became more forceful and the blood pressure rose to 50. The left lung was collapsed, but there was no blood in the pleural cavity. The pericardium, muscles, and skin were closed without drainage. The patient was conscious at the end of the operation. He was given 1,500 c.c. of physiologic salt solution during the operation and two hours later a transfusion of 500 c.c. of blood.

The immediate reaction to the operation was excellent, but forty-eight hours later he developed pneumonia in both lungs and died on the third day.

Necropsy showed that the wound in the heart was healed, and that there was no further hemorrhage or pericardial infection. There was a widespread pneumonia in both lungs.

CASE 7.—J. L., No. 70281, a negro man of twenty-one years was admitted Feb. 23, 1935, at 11:30 P.M. He was rational and stated that he had been stabbed in the

chest about thirty minutes previously. Following the injury he was able to fight for about ten minutes when he collapsed and was brought to the hospital. There had been very little bleeding. There was a wound 3 cm. long through the fifth costal cartilage at the sternal border which was not bleeding nor sucking air. The pulse was weak and irregular and the blood pressure 50/25. The lips and tongue were cyanotic. The neck and arm veins were moderately distended. Operation was performed immediately under nitrous oxide and oxygen anesthesia. The fifth rib was exposed by an incision over it and 8 cm. of rib and cartilage removed. The left pleura was displaced laterally and the pericardium exposed. It was tense and bulging. A pericardial wound was enlarged and the heart began to bleed profusely. Clots and blood were removed from the pericardium. A stay suture of silk was placed in the apex and the heart was lifted forward. A wound 1 cm. long was found near the apex of the right ventricle which was gushing blood with each contraction of the heart. The bleeding was controlled by placing the left index finger over the wound and passing a suture under the finger. Three additional sutures closed the wound. The blood and clots were removed from the pericardial cavity, and the contractions became forceful and regular. The blood pressure rose to 110/70. He was given 1,500 c.c. of physiologic salt solution intravenously during the operation.

Convalescence was without incident. Electrocardiograms made eighteen hours after operation showed an elevated and concave S-T interval, and repeated tracings showed the same condition. He was able to leave the hospital twenty days after operation, and at present is working as a delivery boy, riding a bicycle, without symptoms of cardiac damage.

CASE 8.—W. J., No. 70438, a negro male of thirty-two years, was admitted March 3, 1935. He had been picked up on the street, was unconscious, and no history could be obtained. (It was later learned that after being stabbed in the chest with a sharp-pointed bread knife he walked about 75 feet, when he became exhausted and "noticed that his head was swimming.") There was a stab wound in the second left interspace, 2 cm. long and 1 cm. from the sternal border. It was not bleeding or sucking air. His clothes showed evidence of only slight hemorrhage. The pulse could not be felt. Respiration was labored, and the blood pressure could not be obtained. Heart sounds were not audible with a stethoscope. Immediate operation was done with procaine anesthesia, supplemented later with nitrous oxide and oxygen inhalations. Through a transverse incision 6 cm. of the third left cartilage was removed. The lung was collapsed and the pleura had been opened by the stab wound. There was no blood in the pleural cavity. The pericardium was blue, tense, and bulging. A small wound on its anterior margin was enlarged, and when this was done blood spurted out for a distance of 6 or 8 cm. One hundred and fifty c.c. of blood were aspirated from the pericardial cavity; and the heart, which had been fibrillating, began to contract vigorously. A stay suture was placed at the apex and the heart lifted forward. A wound 1 cm. long was found 2 cm. from the base of the right ventricle. It had definitely entered the right ventricular cavity. It was closed with two sutures of silk, which controlled the bleeding. An attempt was then made to suture the pericardium, but the heart was so dilated that this was impossible. The muscle and skin were closed with interrupted sutures without drainage. He was given 1,000 c.c. of physiologic salt solution during the operation and 400 c.c. of blood immediately afterward. He was conscious at the end of the operation and his pulse was 110, blood pressure 100/65.

Immediate convalescence was good, but on the tenth day he developed subcutaneous emphysema which began at the neck and rapidly spread over his whole body. Aspiration of air from the left chest and the introduction of closed intercostal suction was of no value in checking its progress. He died on the fourteenth day, apparently from mediastinal emphysema. Necropsy was not done.

CASE 9.—L. C., a negro woman of twenty-seven years, was admitted March 30, 1935, at 10:30 P.M. One hour previously she had been stabbed in the chest with a knife. There was a profuse hemorrhage which stopped in a few minutes, and after this she fainted. She had fully regained consciousness when admitted to the hospital, but complained of faintness and weakness. There was a small wound in the fourth interspace near the sternal border, which was not bleeding or sucking air. The neck veins were markedly engorged. The pulse was 100, respirations were 26, blood pressure not obtainable. Operation was done under nitrous oxide and oxygen anesthesia. Through a transverse incision the fifth left cartilage was excised and the fourth cartilage cut and retracted upward. The left lung was collapsed with air, but no opening could be found in the pleura, which was displaced laterally. The pericardium was distended and not pulsating. It was opened and about 100 c.c. of blood removed. The heart then beat vigorously and the blood pressure rose to 110. A wound 1 cm. long in the right ventricle, midway between base and apex, was closed with silk. The pericardium and chest wall were closed with silk without drainage. At the end of the operation the pulse was 100, blood pressure 100/56.

She made a rapid convalescence and was able to leave the hospital on the eighteenth day.

CASE 10.—E. J., No. 74226, a negro man of twenty-four years, was admitted Aug. 31, 1935. He was unconscious and no history could be obtained. There was a moderate amount of blood on his clothes. There was a small stab wound in the second left interspace near the sternum, which was not bleeding and not sucking air. Lips and tongue were pale and cyanotic. The neck vessels were distended. The pulse was weak and irregular, respirations 18, and blood pressure 50 systolic. Under procaine infiltration supplemented with nitrous oxide and oxygen anesthesia, a T-shaped incision was made, and the left second and third cartilages and the upper left portion of the sternum were removed. There was an opening in the pleura, and the left pleural cavity contained about 1,500 c.c. of blood. There was a wound 1 cm. long in the upper part of the pericardium, which was not bleeding. The pericardium was tense and bulging. As soon as it was opened, 200 c.c. of blood was aspirated from it, and the heart, which was fibrillating, began to contract strongly and the blood pressure rose from 50 to 80 systolic. Bleeding was profuse from a wound in the pulmonary artery 1 cm. long and 1 cm. distal to the valves. It was controlled by grasping each side of the wound with Allis forceps and approximating the edges. The wound was closed and the hemorrhage controlled with six interrupted sutures of silk. The pericardium, skin, and muscles were closed without drainage. During the operation the patient was transfused with 1,500 c.c. of citrated blood which was aspirated from his chest and pericardium.

His immediate convalescence was good, but on the second day the temperature rose to 102° F., and he died on the third day after developing definite signs of pneumonia in both lungs.

CASE 11.—M. T., a negro man of thirty-four years, was stabbed in the chest with a knife on March 5, 1933. The wound bled profusely. It was sutured in the out-patient department and he was allowed to return home. There he became unconscious for several hours but recovered. Aside from weakness, which he attributed to loss of blood, he had no further symptoms, and returned to his work as an automobile mechanic three weeks after the injury. Because of shortness of breath he was unable to work and again entered the hospital. At that time (April 3, 1933) there was a scar in the third intercostal space near the sternum which was well healed. Physical examination, confirmed by roentgenogram, showed the pericardial area greatly enlarged. His neck and arm veins were distended. Temperature, pulse, and respirations were normal. The pericardial cavity was aspirated three times, and 500 c.c. of bloody fluid removed on each occasion. Following this

the pericardium returned to its normal dimensions, and he was able to leave the hospital. At the present time he is working as a mechanic and has no cardiac symptoms.

CASE 12.—J. K., a negro man of twenty-two years, entered the hospital on March 29, 1936, at 5 P.M. He walked into the emergency clinic and suddenly collapsed. No history could be obtained but it was later learned that he had been stabbed with an ice pick. The wound was not bleeding, and there was only a small spot of blood on his shirt.

There was a small wound in the third left interspace, 3 cm. from the sternum. The neck and arm veins were distended. The mucous membranes were cyanotic. The pulse was imperceptible, and the heart sounds were distant and slow (about 60 per minute). Blood pressure was 80/60 mm. of mercury, and venous pressure (median basilic) was 250 mm. of water. Examination of the lungs was normal. A diagnosis of cardiac tamponade was made and immediate operation performed under nitrous oxide and oxygen anesthesia.

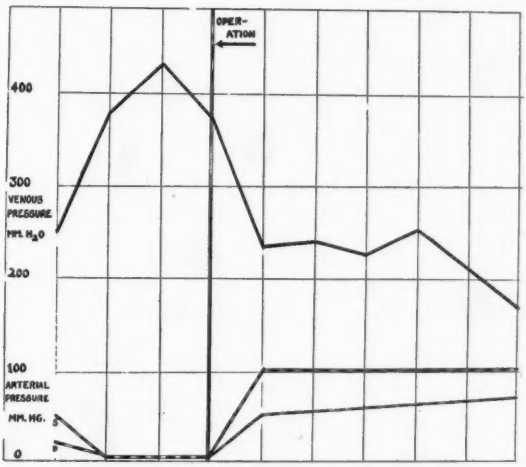


Fig. 3.—The venous and arterial pressures as recorded in Case 12. Venous pressure is given in millimeter of water and arterial pressure in millimeter of mercury.

A T-shaped incision was made over the second left costal cartilage, the overlying skin and muscles were retracted and the second and third cartilages and a portion of the sternum removed. The left pleura was dissected free from its attachment to the sternum and held laterally. The pericardium was tense and not pulsating. At this time the venous pressure had risen to 430 mm. of water. The pericardium was opened between stay sutures, and about 200 c.c. of blood was aspirated. There were no clots in the pericardial sac. The arterial pressure immediately rose to 100 mm. of mercury, and the venous pressure dropped to 230 mm. of water. Blood was seen to well up from the left lateral side of the heart, and by means of a stay suture the heart was rotated medially, and a bleeding point was located on the lateral surface. A branch of the left coronary artery was bleeding, and this was controlled by one suture of silk. The pericardium was loosely closed with interrupted sutures of silk, and the operative wound closed in layers with silk.

He was in good condition at the end of the operation, and the arterial pressure was 100/55 mm. of mercury and the venous pressure 360 mm. of water. The next

morning his condition was satisfactory, and the arterial pressure was 100/65 mm. of mercury and the venous pressure 170 mm. of water. Roentgenogram of the chest was normal except a small pneumothorax on the left side. Twenty-four hours after operation the temperature rose to 106° F., and he died twelve hours later. The cause of death was not known until necropsy was performed, when a pure culture of *Streptococcus hemolyticus* was recovered from the heart's blood. There was no evidence of pericardial infection, and there had been no bleeding. There was a small amount of serosanguineous fluid in each pleural cavity. (Fig. 3 shows the arterial and venous pressure changes in this case.)

CASE 13.—L. M., No. 79112, a negro woman of thirty years, entered the hospital April 14, 1936, at 10 P.M. She was in marked shock, and the blood pressure could not be obtained and the pulse could not be felt. Following saline infusion the blood pressure rose to 100/75. The pulse became strong and about 100 per minute. She gave a history of having been stabbed in the chest with an ice pick shortly before admision and having collapsed about ten minutes later. On examination there were three small punctured wounds on the anterior surface of her chest just to the left of the sternum and in the first and second interspaces. The neck veins were distended and lips cyanotic. Lungs were clear and heart sounds strong and regular. The venous pressure was 225 mm. of water. This rose in thirty minutes to 250 mm. of water. Tamponade of the heart was suspected, and fluoroscopic examination showed practically no cardiac movements. Operation was carried out under nitrous oxide and oxygen anesthesia. The second costal cartilage was excised and the pericardium exposed after dissecting the pleura laterally. The pericardium was tense and blue. It was incised, and 160 c.c. of fluid blood were removed by suction. No bleeding point could be found, but a small amount of blood kept oozing from the region of the left auricle. The pericardium was loosely closed and the skin and muscles were closed without drainage. An opening was made in the left pleura during its dissection, and this was allowed to remain open in order that any bleeding from the pericardium would drain into the left pleural cavity. The convalescence was uneventful except for some difficulty in breathing on the third day. This was relieved by aspirating 300 c.c. of bloody fluid from the chest. At the present time (May 1, 1936) she has apparently recovered without complications.

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# FURTHER DATA ON THE ESTABLISHMENT OF A NEW BLOOD SUPPLY TO THE HEART BY OPERATION*

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I SHOULD like to report briefly upon our work concerning the establishment of a new blood supply to the heart by operative methods. Up to the present time I have operated upon eleven patients with sclerosis of the coronary arteries and angina pectoris. These patients were totally disabled and constituted an extremely bad-risk group for any operative procedure. Six patients are living and five are dead. I shall refer briefly to the results obtained in these patients. A detailed report will be made by Dr. Harold Feil and myself. Dr. Feil selected the patients for operation.

The first patient was a farm laborer of forty-eight years, who had typical substernal pain on effort of two years' duration. He finally was forced to give up his work because of pain. Operation was done February 13, 1935. The patient claims that he has been cured by the operation. He has no pain and he takes no medication. He has gained weight. He is doing physical labor; he shoveled snow during the past winter.

The second patient was an executive of fifty-one years. He developed angina pectoris in 1929. He was forced to retire in 1933. A total thyroidectomy was done in 1934. He kept his basal metabolic rate at about minus 20. When he increased the dosage of thyroid extract, the pain became more acute. He was bedridden and had no interest in life. In June, 1935, I operated to increase the blood supply to the heart. He feels that he has been greatly benefited by the operation. He is able to take three times as much thyroid extract as he took before operation, and he maintains a normal metabolic rate. He experiences a tightening over the precordium but claims that he has no real anginal pain. He is still weak, but he is up and about ten hours a day, and he has some interest in life. This patient may have degenerative changes or fibrosis in the heart of a permanent nature. He feels that the operation of cardiac anastomosis was decidedly worth while.

The third patient was a surgeon of fifty years. He had had angina pectoris for five years. He had been completely disabled for three and one-half years. He probably had had a thrombosis of a coronary artery one and one-half years before the operation on the heart was done. This

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patient also had sclerosis of the femoral arteries and diabetes mellitus. He claims that the operation has helped him. He believes the attacks of pain are less frequent and less severe than they were before operation.

The fourth patient was a machinist of fifty-five years. He had typical angina of effort of two years' duration, and had been unable to work for five months. He had generalized arteriosclerosis. The vessels of the legs were markedly sclerotic. Operation to increase the blood supply to the heart was done August 31, 1935. The patient claims that he is entirely relieved of pain since the operation. He returned to his former job as a machinist and has been working steadily for the past four months. His tolerance for exercise, as determined by means of the step test, is limited not by any precordial discomfort, because he has none, but by the sclerosis of the femoral arteries.

The fifth patient was a man of forty-six years who had had typical attacks of anginal pain for three years. He had to give up his work as a painter. Operation was done January 6, 1936. He has made a slow convalescence from the operation. Four months have elapsed and only slight improvement in his condition has taken place. He occasionally has anginal pain. The result in this patient is questionable.

The sixth patient was forty-two years old. He had anginal pain for sixteen months and became totally incapacitated. He also has sclerosis of the arteries of the legs. He was operated upon on February 26, 1936. This patient claims that he is entirely free from pain and that the operation has been highly beneficial to him. His tolerance for exercise has increased.

The five deaths were as follows: The first patient developed thrombosis at the bifurcation of the aorta due to a calcified ulcer of the aorta. This occurred on the sixth day after operation and the patient died a few hours later. The heart showed extensive sclerosis of all coronary arteries and extensive fatty degeneration and fibrosis of both ventricles. The second patient developed thrombosis of the middle cerebral artery, became unconscious, developed pneumonia, and died three days after operation. The third patient developed a gas bacillus mediastinitis and died two days after operation. The fourth patient died suddenly from a coronary attack twenty-four hours after operation. The fifth patient died suddenly one day after operation. He had an aneurysm of the left ventricle on the posterior aspect of the heart. Every one of these cases showed advanced sclerosis of the coronary arteries, and two of them showed extensive and permanent degenerative changes in the myocardium.

The operative procedure as applied to the human patients has been modified as we have gone along. In the first patient the insertion of the left pectoral muscle was cut, and the entire muscle was therefore sacrificed. In the next patient a bilateral approach to the heart was made, and

the inferior portions of the right and left pectoral muscles were grafted upon the heart. The bilateral H-shaped incision, which I described in 1931,[1] was changed to a transverse incision extending from one nipple to the other. In an attempt to reduce the magnitude of the operation a unilateral transverse incision was then developed, and grafts from the inferior portion of the left pectoral muscle were used (Figs. 1, 2, and 3). The tissues available to the heart for a blood supply are the pectoral muscle, the triangularis sterni muscle, the intercostal muscle, the inter-

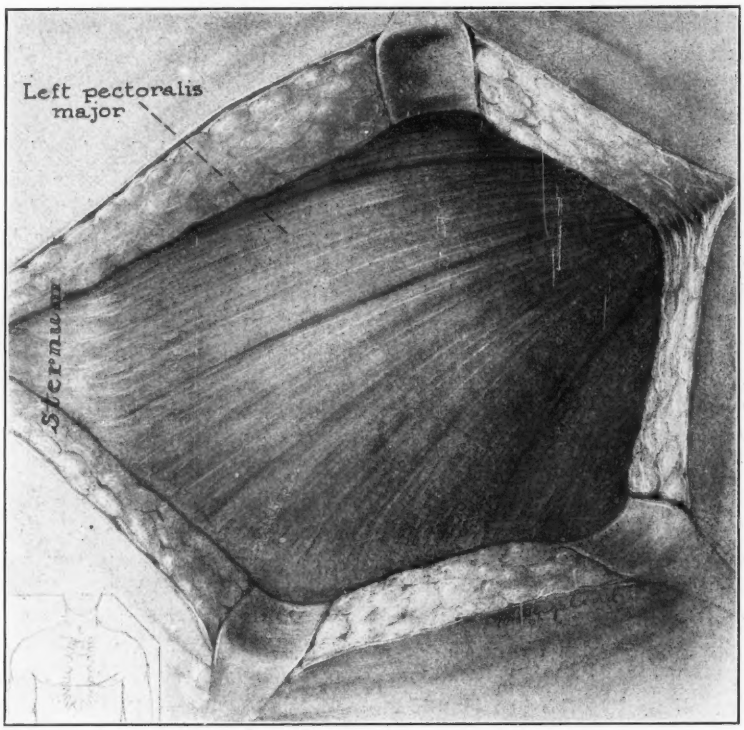


Fig. 1.—The transverse skin incision is shown in the inset. Flaps of skin and subcutaneous fat are dissected from the pectoral muscle. The inferior portion of the left pectoral muscle is dissected from the underlying ribs. The fibers of the muscle are cut across laterally, and a broad graft of muscle is made with its base attached along the sternum. This graft receives its blood supply from the internal mammary and epigastric arteries. (Figs. 1-3, from Levy, *Coronary Disease*. By permission of The Macmillan Co., Publishers.)

costal vessels, the internal mammary vessels, subcutaneous fat, mediastinal fat and the parietal pericardium. I have been asked whether we used omentum. In our early experiments, four years ago, omentum was brought up through an opening in the diaphragm and grafted onto the heart. I have not used it on human patients because it adds certain complicating features to the operation which I shall not discuss.

One important development that this work must take is to adapt the operation to the patient. The procedures that I have carried out in the eleven patients with coronary sclerosis are not operations of great magnitude for the average patient. Patients with sclerosis of the coronary arteries are notoriously bad risks for any kind of operative procedure, and this statement is especially applicable to the eleven patients who had this operation performed. They were advanced cases. But patients

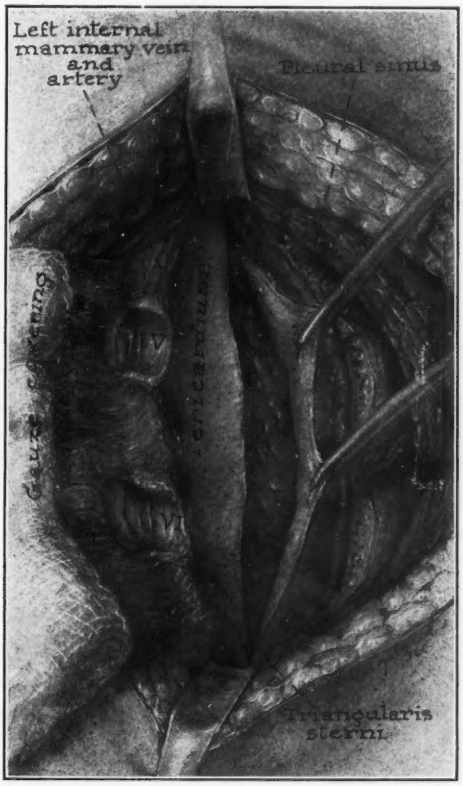


Fig. 2.—The sixth and fifth (and sometimes the fourth) costal cartilages are removed. The pericardium is widely incised, and the epicardium and lining of pericardium are roughened by mechanical methods. Special burs were devised for this purpose.

with advanced coronary sclerosis are not to be deprived of any benefit that this surgical principle has to offer them, and the operation has to be adapted to their capacity to withstand it.

Two developments are being carried out. One concerns the reduction in irritability of the heart at the operating table by the application of drugs to the surface of the heart. This study has been made by Dr.

Frederick R. Mautz and is being reported separately. Extrasystoles can be reduced, auricular fibrillation can be stopped, the ventricular rate can be slowed, and the danger of ventricular fibrillation at the operating table can be avoided by this development. It will find a place in all cardiac surgery.

The other development consists in making a vascular bed available to the myocardium by a much more simple type of operation. Destruction

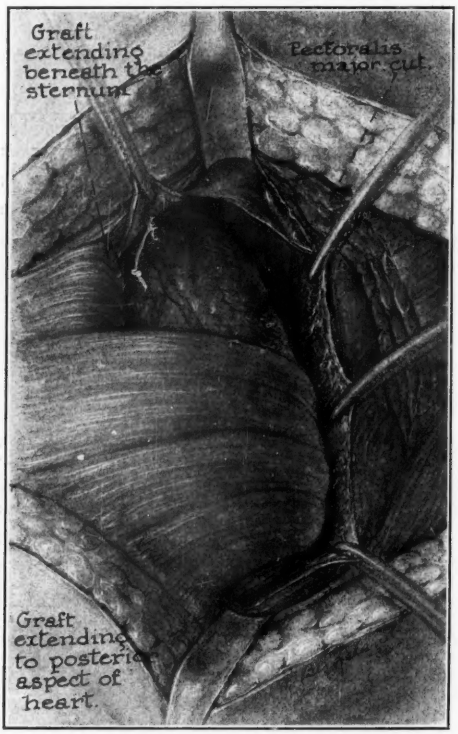


Fig. 3.—The major portion of the graft is then swung around to come into contact with that part of the myocardium supplied by the circumflex branch of the left coronary artery. It is anchored to the parietal pericardium. The smaller portion of the graft is placed beneath the sternum and in contact with the right ventricle. Sutures are not placed in the heart. The pericardium is left widely open. The flaps of subcutaneous fat and skin are sutured, and the wound is closed.

of epicardium and mesothelial lining of pericardium is dangerous if carried out by the use of chemical agents. The mediastinal tissues are very susceptible to chemical trauma, and compression scars may develop after the use of chemicals.[2] We have been stripping off the epicardium by means of burs, but this method also is not without danger. Stripping off the epicardium produces many extrasystoles. We should also like to avoid cutting and placing grafts of skeletal muscle on the heart. These

large muscle grafts may be a factor in the development of arterial thrombosis by the liberation of tissue juices into the blood stream.* Thrombosis was the cause of death in three of our cases. In the human being a deposit of fat usually exists in the mediastinum. The parietal pericardium is surrounded by a layer of fat several millimeters in thickness. We have used this fat successfully as a vascular bed in experiments upon animals, and we believe it will work out satisfactorily in human patients with advanced coronary sclerosis. Our next problem is to establish anastomoses between the mediastinal fat and the myocardium. Dr. Mautz and I have been working on this problem, and it seems that a relatively bland foreign body, such as powdered bone, placed between the heart and the fat will be satisfactory. The reaction produced by the bone brings about the development of blood vessels. We are ready to apply this method to human patients. I believe it will prove to be applicable especially to the bad-risk patient. The procedure in itself should carry with it little risk. Under procaine anesthesia the left fourth costal cartilage is removed. The parietal pericardium is opened, and the incised margin is sutured to the fascia of the pectoral muscle. Powdered bone is introduced into the pericardial cavity and spread around over the ventricles. The fat and skin are closed without drainage. If the heart becomes compressed by the formation of fluid, the fluid can be removed without difficulty by aspiration.

Further investigation may give us an injection method for the establishment of a vascular bed. We have used many different solutions with this purpose in mind, but none has proved to be satisfactory. There are a number of considerations that cannot be overlooked before such a method is acceptable, and no attempt should be made on a human patient without adequate experimental trial.

I shall give a brief résumé of the experimental work to date. In our first series of experiments[3] with Dr. V. L. Tichy the mesothelial envelope around the heart was destroyed by roughening the lining of the parietal pericardium and also the epicardium by means of a bur. A new vascular bed was constructed by bringing various grafts onto the myocardium. The tissues that were used for the new vascular bed were fibrous pericardium, pericardial fat, pedicle grafts of skeletal muscle from the chest wall, and omentum brought up through an opening in the diaphragm. Anastomoses between the grafts and the heart could be demonstrated in three weeks after the grafts were placed. Anastomoses increased after a physiologic need for blood was established in the myocardium. The physiologic need for blood is perhaps more accurately defined as a pressure differential between the two vascular beds. It was produced by occlusion, partial or complete, of major coronary arteries.

*This suggestion was made to me by Dr. W. H. Howell. We contemplated the use of purified heparin to prevent clotting after operation.

Anastomoses were demonstrated in two ways. They were demonstrated by injection of dye into the myocardium by way of the grafts. This was done by excluding every portal of entry into the heart except the extracardiac anastomoses. The aorta was clamped. It was cut between clamp and heart so that injection through coronary arteries and thebesian vessels was impossible. Dye was then injected into the aorta. A profuse and deep staining of the myocardium was obtained in specimens in which a physiologic need for blood existed. A capillary injection of myocardium was obtained.

Anastomoses were also demonstrated by injecting a solution of barium sulphate into a coronary artery and tracing the solution in the chest wall by means of x-ray pictures. The solution of barium sulphate had the consistency of thick cream. It was too thick to enter capillaries. The experiment was carried out as follows: The heart was given a collateral vascular bed, and a physiologic need for blood was produced by occlusion of the right coronary artery. Several months later the left coronary artery was injected. X-ray examination showed the opaque mass in both right and left coronary arteries. From the heart it spread to the chest wall and filled internal mammary, many intercostal, diaphragmatic, and epigastric arteries.

In some of the experiments the heart was given a collateral vascular bed, and at the same operation partial occlusion of one of the coronary arteries was produced by means of a silver band. Several weeks later this artery was completely occluded. The right coronary artery or either major branch of the left coronary artery could be occluded, with recovery following regularly without infarction if the occlusion was done in two stages.

In another series of the experiments the heart was given a collateral vascular bed, and then occlusion of the coronary arteries was carried out in four or five subsequent operations. It was possible to occlude completely the right and both major branches of the left coronary arteries. The entire coronary supply was cut off except for the septal branch which comes off above the bifurcation of the left. This was accomplished with recovery and without infarction.

In other experiments the heart was given a collateral vascular bed, and several weeks later the right coronary artery was ligated completely in one stage. There were four deaths and eight recoveries. The right coronary artery was ligated in normal dogs for controls; in these control experiments there were seven deaths and three recoveries. It appears that the vascular grafts protect the heart against sudden complete occlusion of the right coronary artery. In this respect the grafting is prophylactic. A similar statement can be made concerning ligation of the ramus descendens of the left coronary artery.

Two types of experiment are presented for comparison: (A) Complete ligation in one stage of four or five peripheral branches of the

coronary arteries over the apex of the left ventricle was always fatal; ventricular fibrillation developed. (B) The right and both major branches of the left coronary arteries could be occluded by about one-third of their cross-section in one stage with recovery. The total amount of blood going through the coronary system in A can be assumed to be greater than in B; yet A was a fatal experiment and B resulted in recovery. These experiments bring out an entirely new point so far as the heart is concerned, namely, the importance of an equal distribution of blood to the myocardium. Total coronary blood flow is one factor that concerns the preservation of the heartbeat, but distribution of blood flow is another important factor.

The collateral vascular bed can function in effecting an equal distribution of blood to various parts of the myocardium. This is effected in two ways: first, by bringing blood from extracardiac sources into the myocardium; and, second, by transporting blood from one part of the heart where the circulation is good to another part of the heart where the circulation is deficient. In the latter respect the grafts act as anastomotic bridges connecting the beds of different vessels.

The grafted tissues do not interfere with the movements of the heart. Cardiac adhesions are usually silent and incidental lesions. A special study of this subject has been carried out by Dr. Robert M. Hosler and Dr. John E. Williams. This work is reported separately.

ADDENDUM:

Since this article was written, powdered bone has been used in two patients who were extremely bad operative risks.

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# REDUCTION OF CARDIAC IRRITABILITY BY THE EPICARDIAL AND SYSTEMIC ADMINISTRATION OF DRUGS AS A PROTECTION IN CARDIAC SURGERY*

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## INTRODUCTION

RECENT advances in cardiac surgery[1, 2, 3] reopen questions regarding the pathologic physiology of the heart and circulation. From the viewpoint of the surgeon an operation upon the heart is vastly different from an operation upon any other organ, not only from a mechanical point of view but also because of the factors which disturb normal function during the operative and postoperative periods. If a cardiac operation is to be successful, the heart must be able to withstand the operative trauma, participate in the postoperative reaction, and at the same time maintain an adequate circulation of blood.

## OBSERVATIONS ON DISTURBANCES OF RHYTHM

This report concerns a study of the disturbances of cardiac rhythm associated with cardiac surgery and an experimental investigation of methods for the prevention and treatment of these disturbances. This work was done in connection with experimental studies of collateral coronary circulation and ligation of coronary arteries in dogs. Suggestions for clinical application are based upon the observations made upon a group of patients subjected to cardiac operations.

Many disturbances of cardiac rhythm have been noted associated with cardiac operations, as indicated by clinical observation and numerous electrocardiographic studies. Some of these disturbances were of a serious nature and occasionally the immediate cause of a fatality. One or more of the following disturbances of rhythm were noted in every cardiac operation:

1. Sinus tachycardia
2. Heart-block
3. Extrasystoles
    a. Induced by the operator
    b. Spontaneous
4. Ventricular tachycardia
5. Auricular fibrillation
6. Ventricular fibrillation

*From the Laboratory of Surgical Research, the Western Reserve University School of Medicine.

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DISTURBANCES OF RHYTHM IN EXPERIMENTAL STUDIES

In the experimental animals sinus tachycardias with rates of 180 per minute or more have been frequently encountered at operation. While this rapid rate, maintained for only a short time, may not be harmful, it may make a delicate dissection very difficult on account of the rapid heart motions. Extrasystoles occur frequently due to mechanical stimulation of heart muscle during the nonrefractory phase of the cardiac cycle and are also seen to originate spontaneously at times when the heart is not being traumatized. These spontaneous extrasystoles are seen most frequently after there has been considerable trauma to the surface of the heart and particularly after coronary artery ligation. Occasional extrasystoles cause little disturbance of the circulation, but it has been shown that a stimulus introduced just at

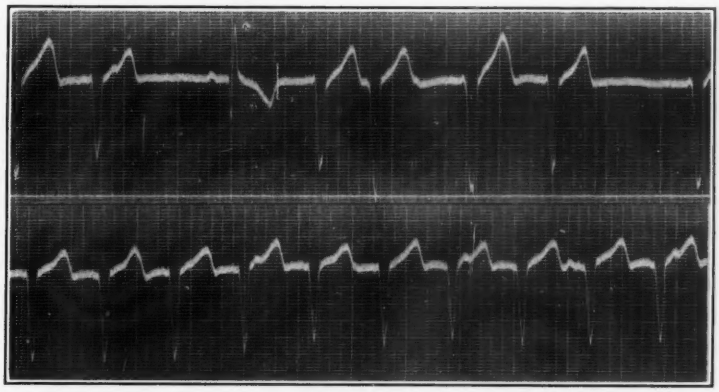

Fig. 1.—Extrasystoles forty-eight hours following an infarct of the left circumflex area. The upper record shows many extrasystoles arising from various foci in the infarcted zone and in addition one normal complex. Below is a record from a paroxysmal ventricular tachycardia in which the focus of impulse formation is localized to a much smaller zone.

the end of systole in the partially refractory phase of the cardiac cycle is likely to cause ventricular fibrillation.[4] An ischemic zone of myocardium frequently becomes a focus for the formation of ectopic beats. Lewis[5] has pointed out that acute paroxysmal ventricular tachycardias frequently follow the ligation of the right coronary and descending ramus of the left coronary arteries in the dog. In two-thirds of his experiments ventricular fibrillation developed within from a few minutes to three hours as a result of ligation of the right coronary artery near its origin. We have observed the development of ventricular tachycardia frequently after partial or complete obstruction of a major coronary artery (Fig. 1). In general, ligation of either branch of the left coronary artery led to left ventricular extrasystoles with a downward initial deflection in the electrocardiogram, and ligation of the

right coronary artery led to right ventricular extrasystoles with an upright initial deflection in the electrocardiogram. Frequently an animal would appear to be in good condition twenty-four hours after a coronary ligation, then suddenly die, presumably owing to sudden development of ventricular fibrillation. Such accidents occurred frequently during or shortly after the eating of a small meal. If an animal continued to survive, the ventricular tachycardia frequently persisted three to four days, after which a normal mechanism supervened.

Auricular fibrillation has developed occasionally during an operation when there was considerable traction on an auricular appendage in order to get exposure in the region of the point of origin of a coronary vessel. This has usually occurred in the longer and more difficult operations and has almost always been followed by ventricular fibrillation within a few minutes.

Various degrees and types of heart-block have also been noted following coronary artery ligation. Delayed conduction and dropped ventricular complexes were frequently noted months after a coronary artery occlusion in animals apparently in excellent health.

### DISTURBANCES OF RHYTHM IN HUMAN SUBJECTS OF HEART OPERATIONS

In fourteen human subjects of heart operations, performed by Dr. C. S. Beck in the Lakeside Hospital, the same group of disturbances was encountered. Numerous extrasystoles occurred in all cases when the heart was being retracted or touched with instruments, due to stimulation of the myocardium. During rest periods in the course of the operation, spontaneous extrasystoles, singly and in short runs, have frequently been noted, especially in those cases in which a pericardial scar was being resected. These patients also generally had rapid heart rates during the operation and the early postoperative period.

Two patients developed rapid arrhythmias a few hours after the operation. In one patient the electrocardiogram showed auricular fibrillation with numerous premature ventricular beats occurring singly and in short runs. In the other patient the irregularity subsided without specific therapy, and no electrocardiographic studies were obtained during the attack which was thought to be auricular fibrillation. Three patients showed numerous premature ventricular systoles in daily electrocardiograms during the early postoperative period. Two patients who had been in fair condition postoperatively died quite suddenly. They had been quite clear mentally and suddenly complained of anginal pain which was immediately followed by gasping, pallor and death, indicating that the circulation of blood had ceased while the respiratory center was still capable of responding. These deaths are believed to have been due to the onset of ventricular fibrillation. There were no data to indicate the frequency of ventricular

extrasystoles preceding the ventricular fibrillation in these cases, but no marked arrhythmia was present. Autopsy showed no recent coronary thrombosis, only severe obliterative coronary sclerosis.

One patient with chronic cardiac compression showed many premature auricular and ventricular beats while the scar was being resected and developed ventricular fibrillation on the operating table.

The question is then raised: Are there any means at our disposal to make the heart less likely to develop these disturbances of rhythm? There are two avenues of approach: It might be possible for the surgeon to apply to the epicardium a drug which would reduce the irritability of a surface layer of myocardium and eliminate many of the extrasystoles at the operation. Also it might be possible to give a drug systemically which would lessen the danger of the development of auricular or ventricular fibrillation.

## EXPERIMENTS TO DETERMINE THE EFFECT OF DRUGS ON SURFACE EXCITABILITY

A simple method of demonstrating the effect of a drug applied to the epicardium upon the underlying myocardium is to record an electrogram from a point on the surface of the heart before and after the application of a drug. W. Straub,[6] many years ago, pointed out the value of this method of studying specific drug action on the myocardium. A drug which is absorbed through the epicardium and affects the underlying myocardium so as to modify its function will cause a change in the action current. Von Werz[7] has made an analysis of the possible types of change which one should theoretically be able to produce. He concluded that any factor which would change either the *intensity* or the *duration* of the electrical activity under the exploring electrode would distort the electrogram in a characteristic fashion resulting in the well-known monophasic type of injury current, Fig. 2.

Wiggers[8] has shown that solutions of ionizable potassium salts painted on the epicardium produce a monophasic type of action current in the standard electrocardiogram identical to the record obtained in an acute coronary artery occlusion. Furthermore, the depressant action of ionized potassium on myocardial irritability is well known, and since the electrocardiographic studies indicate that it is readily absorbed when applied to the epicardium, solutions containing ionized potassium salts might be of value in reducing surface irritability of the heart.

In view of the fact that the members of the cocaine group are very readily absorbed from mucous membranes and have also been shown to have some effect on myocardium,[9] members of this group were studied and found to produce monophasic types of action currents very readily, both in the direct leads from the epicardium over the area to which the drugs had been applied, and also in the standard Lead II when the drugs were allowed to diffuse through the entire pericardial cavity.

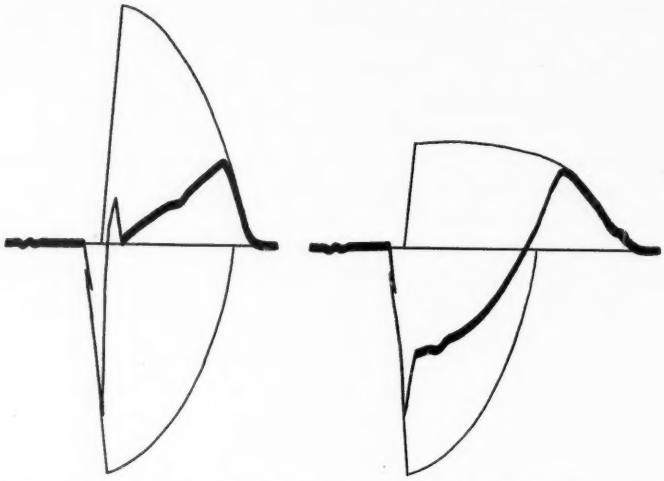

Fig. 2.—The electrograms of Fig. 3 are reconstructed to show the components of negativity under each of the two electrodes. Procaine on the test area has led to a reduction in intensity and increase in the duration of the component of negativity under the exploring electrode leading to a monophasic type of deformation of the electrogram as indicated on the right.

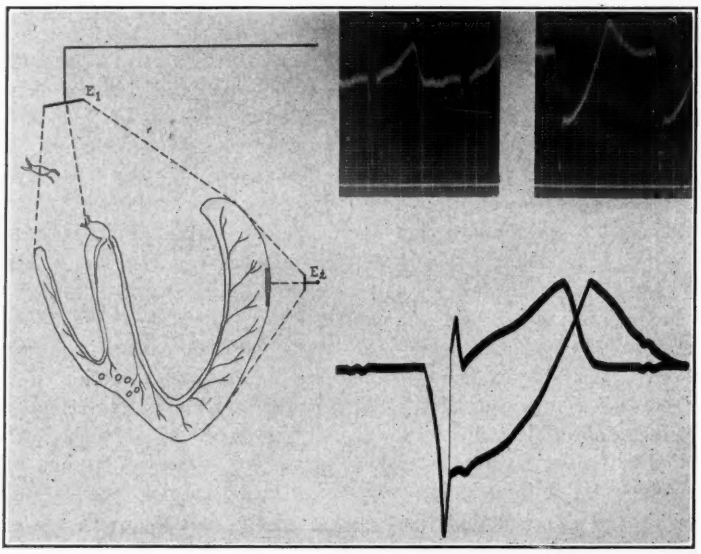


Fig. 3.—The two records at the upper right are segments of electrograms taken from a dog's heart with nonpolarizable electrodes situated as indicated in the diagram at the left, a small pledget of cotton being placed between $E_2$ and the epicardium. The first record is the normal control, and the second was taken one minute after the application of a cotton pledget containing 1 c.c. of 10 per cent procaine to the area for three minutes. Records taken during the following ten minutes showed a gradual disappearance of the monophasic component. Records taken from other areas of epicardium showed no significant change at the time a marked injury component was present in the lead from the procaine area. Below, the two complexes are reconstructed superimposed.

Metycaine, cocaine, and procaine solutions were all found to be effective in producing a local injury current of short duration. The quantity of drug used was in each case sufficiently small that there were no general toxic effects. There was practically no effect on mean blood pressure recorded during the experiments by means of a mercury manometer with a cannula in the femoral artery. Fig. 3 shows an injury current produced by the application of 10 per cent procaine hydrochloride to a small area of epicardium on the left ventricle. Electrograms from a control area to which no drug was applied show no significant change, indicating that a local surface effect can be produced by the drug.

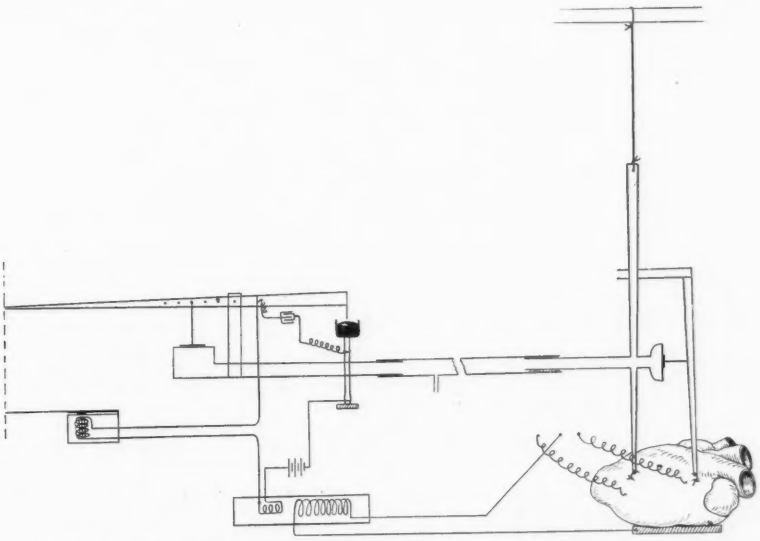

Fig. 4.—The automatic cardiac stimulator. The leads from the secondary coil are so arranged that the pin electrode becomes negative with the break. Extrasystoles are recognized (1) by direct observation of the heart, (2) by observing the compensatory pause on the tambour record, and (3) by the momentary fall in mean blood pressure. The latter is the best indicator for extrasystoles that nearly coincide with the next regular systole. The condenser across the mercury switch prevents arcing.

In order to get some quantitative data on how much reduction in surface excitability can be produced by these drugs we used an automatic cardiac stimulator,[10] shown in Fig. 4. With this arrangement the primary circuit of the induction coil is closed as the cardiac systole shortens the distance between the two levers of the myocardiograph, and the electrode on the recording tambour is forced into the mercury cup. When relaxation occurs, the contact is broken and a break stimulus is delivered to one of the pin electrodes in the epicardium. In this way the position of the secondary coil can be varied so that

that position can be determined at which an extrasystole will *just be produced by the break shock.*

We found with this arrangement that the minimal stimulus necessary to produce an extrasystole was increased after the local application of cocaine, metycaine (Fig. 5), procaine, and potassium chloride solutions to the epicardium around the stimulating pin. The application was made by saturating a small pad of cellucotton, placed around the test point, with the drug and allowing several minutes for the drug to be absorbed. Then the pad was removed, the surface irrigated with saline, and the minimal stimulus necessary to produce an extrasystole redetermined at short intervals.

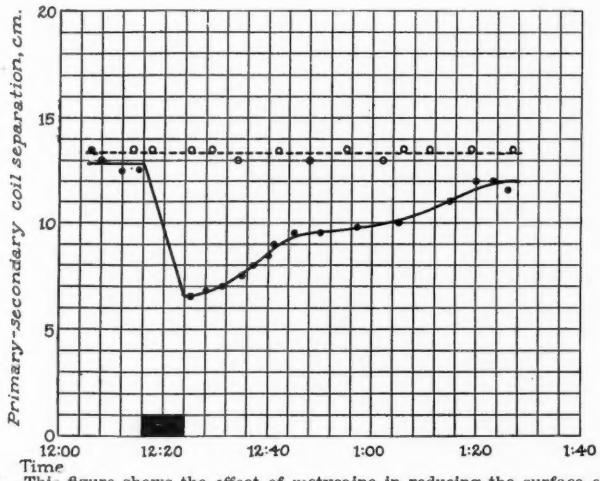


Fig. 5.—This figure shows the effect of metycaine in reducing the surface excitability at a test-point on the surface of the left ventricle as indicated by the reduction of the distance between the primary and the secondary coils necessary just to produce extrasystoles. The dotted line represents results from stimulation of a control point on the left ventricle and shows no change in excitability.

Interpreting the reduction of irritability on the basis of the decrease in coil separation does not give a good idea of the quantitative increase of the energy necessary to produce an extrasystole, since the strength of the stimulus is not related in a linear fashion to the coil separation.

An attempt was made to calibrate the induction coils and to get at least a rough quantitative measure of the relative energy of the break stimulus for various positions of the secondary coil. For this purpose a string galvanometer of a Cambridge electrocardiogram was utilized to record the form of the stimuli, only a small fraction of the stimulus being allowed to pass through the galvanometer, shunts of from 0.10 to 20.0 ohms being placed across the secondary coil. In this way the relative heights of the string deflections for various settings of the secondary could be compared.

Calibration curves were constructed from comparisons of the make stimuli, since the rise from zero to maximum potential occurs more slowly than in the case of the break stimuli; hence the *string* should follow the make stimulus better than the break stimulus.[11] Dr. A. C. Seletzky of the Electrical Engineering Department of the Case School of Applied Science recorded the wave forms of the make and break stimuli with the cathode ray oscillograph. Measurement of the height of make and break deflections showed that the ratio of make to break was constant throughout the range employed in the experiments, provided the conditions in primary and secondary circuits were kept constant, a condition also met in the experiments. By this method the calibration curves for two induction coils were obtained and are given in Table I and Fig. 6.

TABLE I

CALIBRATION DATA FOR INDUCTION COIL USED IN THE REPORTED EXPERIMENTS

| PRIMARY-SECONDARY COIL SEPARATION IN CM. | RELATIVE MAXIMUM DEFLECTION OF MAKE STIMULUS |
|---|---|
| 0 | 100.0 |
| 1.0 | 95.5 |
| 2.0 | 87.6 |
| 3.0 | 78.1 |
| 3.5 | 70.0 |
| 4.0 | 61.0 |
| 4.5 | 51.8 |
| 5.0 | 43.8 |
| 5.5 | 34.5 |
| 6.0 | 26.2 |
| 6.5 | 19.2 |
| 7.0 | 13.8 |
| 7.5 | 9.60 |
| 8.0 | 6.90 |
| 8.5 | 5.00 |
| 9.0 | 3.40 |
| 9.5 | 2.60 |
| 10.0 | 2.01 |
| 11.0 | 1.54 |
| 12.0 | 1.20 |
| 13.0 | 0.96 |
| 13.5 | 0.86 |

Transposing the data from secondary coil settings to the calibration figures gives at least a rough quantitative measure of the amount of reduction of the surface irritability. In Tables II, III, IV, and V and Figs. 7 and 8 are presented the data on experiments with cocaine, metycaine, procaine, and potassium chloride. These data correspond closely with the duration of the monophasic component of the action current previously discussed.

So far then, we have demonstrated a specific myocardial action of several drugs which are readily absorbed from the epicardial surface and which will produce a surface layer of myocardium that will not

give rise to extrasystoles when stimulated. Concentrated solutions of these drugs have been used so that only a very small amount of solution would have to be applied to a small area of epicardium and at the same

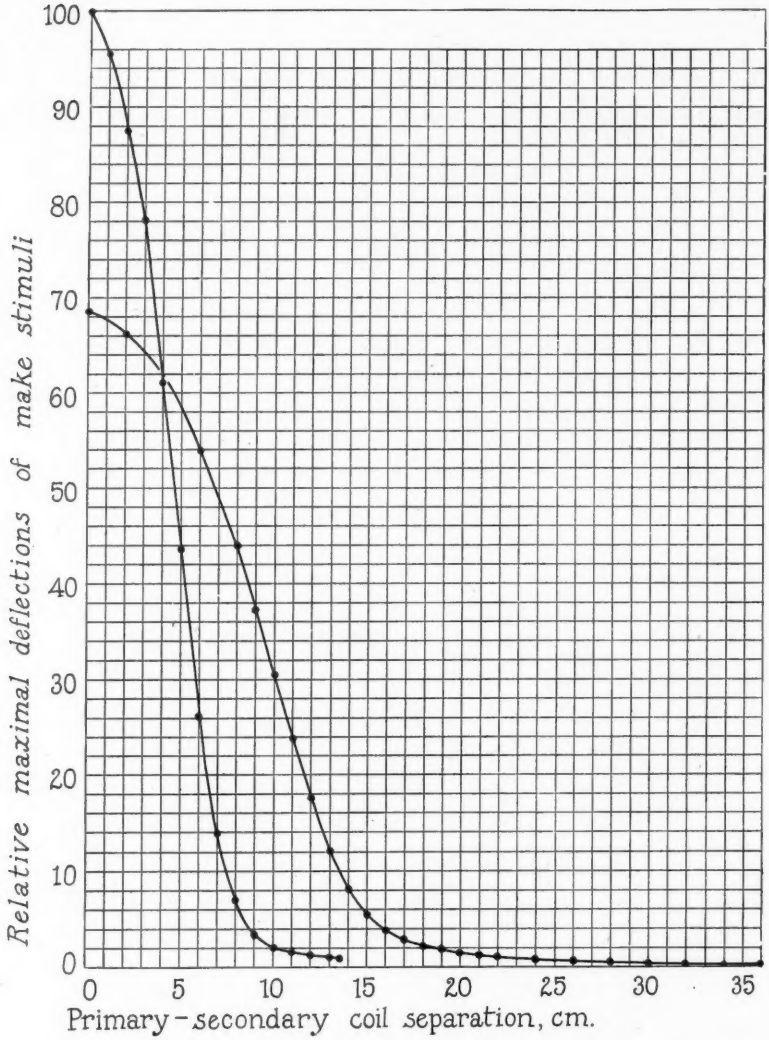


Fig. 6.—Calibration curves for two induction coils determined by comparing the heights of make deflections of a string galvanometer for various settings of the secondary coils. One, the curve for a small short coil with iron core; the other, the curve for a considerably longer coil, also with an iron core.

time have an adequate dose of the drug present to produce the maximal action on the surface myocardium. The optimum concentration to pro-

duce the desired effects and the total dose that can be used in safety are yet to be accurately determined. In the following section will be given the figures which have been found satisfactory in our work up

TABLE II

METYCAINE ON EPICARDIUM

EXPERIMENT 35-208, MALE POLICE DOG, 14.8 KG., BARBITAL ANESTHESIA

| CONTROL AREA BASE LEFT VENTRICLE | | | METYCAINE AREA APEX LEFT VENTRICLE | | |
|---|---|---|---|---|---|
| TIME | COIL SEPARATION | STRENGTH OF STIMULUS CALIBRATION UNITS | TIME | COIL SEPARATION | STRENGTH OF STIMULUS CALIBRATION UNITS |
| 11:53 | 13.5 | 0.86 | 12:06 | 13.5 | 0.86 |
| 12:08 | 13.0 | 0.96 | :12 | 12.5 | 1.12 |
| :14 | 13.5 | 0.86 | :14½ | 12.5 | 1.12 |
| | | | 12:16-12:24 | 10% metycaine to area | |
| :17½ | 13.5 | 0.86 | :25 | 6.5 | 19.2 |
| :25 | 13.5 | 0.86 | :28 | 6.75 | 16.5 |
| :29 | 13.5 | 0.86 | :31 | 7.0 | 13.8 |
| :34 | 13.0 | 0.96 | :35 | 7.5 | 9.60 |
| :42 | 13.5 | 0.86 | :37 | 8.0 | 6.90 |
| :48 | 13.0 | 0.96 | :40 | 8.5 | 5.00 |
| :55 | 13.5 | 0.86 | :41 | 9.0 | 3.40 |
| 1:02 | 13.0 | 0.96 | :45 | 9.5 | 2.60 |
| :06 | 13.5 | 0.86 | :50 | 9.5 | 2.60 |
| :11 | 13.5 | 0.86 | :57 | 9.75 | 2.30 |
| :19 | 13.5 | 0.86 | 1:05 | 10.0 | 2.01 |
| :27 | 13.5 | 0.86 | :15 | 11.0 | 1.54 |
| | | | :20 | 12.0 | 1.20 |
| | | | :23 | 12.0 | 1.20 |
| | | | :26 | 11.5 | 1.54 |

TABLE III

COCAINE ON EPICARDIUM { 5% COCAINE HCl / ¼% $NaHCO_3$

EXPERIMENT 35-145, MALE POLICE DOG, 21.5 KG., INTRATRACHEAL ETHER ANESTHESIA

| CONTROL AREA | | | COCAINE AREA | | |
|---|---|---|---|---|---|
| TIME | COIL SEPARATION | STIMULUS STRENGTH | TIME | COIL SEPARATION | STIMULUS STRENGTH |
| 2:54 | 11.0 | 1.54 | 2:48 | 11.5 | 1.32 |
| 3:04 | 11.0 | 1.54 | 3:02 | 11.5 | 1.32 |
| 3:13 | 11.5 | 1.32 | 3:06-3:09 | 5% cocaine to area | |
| 3:29 | 11.5 | 1.32 | 3:10 | 7.5 | 9.6 |
| | | | 3:14 | 8.5 | 5.0 |
| 3:39 | 11.5 | 1.32 | 3:17-3:20 | 5% cocaine to area | |
| | | | 3:21 | 6.5 | 19.2 |
| | | | 3:32 | 8.5 | 5.0 |
| | | | 3:37 | 11.0 | 1.54 |

to the present time but which without doubt will be subject to some modification as a result of added experience. However, the principles for the application of the drugs to reduce surface excitability of the heart have been established.

EFFECTS OF COCAINE GROUP ON CARDIAC NERVES

The question was immediately raised as to what effect drugs of this group will have on the vagus and sympathetic innervation to the heart when the drugs are allowed to come in contact with the entire pericardial surface.

TABLE IV

PROCAINE ON EPICARDIUM

EXPERIMENT 35-209, MALE POLICE DOG, 23.0 KG., BARBITAL ANESTHESIA

| CONTROL AREA | | | PROCAINE AREA | | |
|---|---|---|---|---|---|
| TIME | COIL SEPARATION | STIMULUS STRENGTH | TIME | COIL SEPARATION | STIMULUS STRENGTH |
| 9:44 | 11.5 | 1.32 | 9:445 | 10.5 | 1.77 |
| 9:46 | 11.0 | 1.54 | 9:46 | 10.0 | 2.01 |
| 9:53 | 11.0 | 1.54 | 9:53 | 10.5 | 1.77 |
| 9:57 | 12.0 | 1.20 | 9:57 | 11.0 | 1.54 |
| 10:00 | 12.0 | 1.20 | 10:01 | 11.0 | 1.54 |
| 10:14 | 12.0 | 1.20 | 10:04-10:11 | 15% procaine applied to area | |
| 10:23 | 13.0 | 0.96 | 10:13 | 7.25 | 11.6 |
| 10:25 | 13.0 | 0.96 | :16 | 7.50 | 9.60 |
| 10:33 | 13.0 | 0.96 | :23 | 9.00 | 3.40 |
| 10:47 | 13.0 | 0.96 | :25 | 9.00 | 3.40 |
| | | | :32 | 10.5 | 1.77 |
| | | | :46 | 11.0 | 1.54 |
| | | | :50 | 11.25 | 1.42 |

TABLE V

POTASSIUM CHLORIDE ON EPICARDIUM

EXPERIMENT 35-209, MALE POLICE DOG, 23.0 KG., BARBITAL ANESTHESIA

| CONTROL AREA | | | POTASSIUM CHLORIDE AREA | | |
|---|---|---|---|---|---|
| TIME | COIL SEPARATION | STIMULUS STRENGTH | TIME | STIMULUS STRENGTH | COIL SEPARATION |
| 12:04 | 13.0 | 0.96 | 12:01 | 10.75 | 1.90 |
| :08 | 13.0 | 0.96 | :02 | 11.00 | 1.54 |
| :18 | 12.5 | 1.12 | :07 | 12.00 | 1.20 |
| :23 | 12.5 | 1.12 | 12:10-12:16 | 10% KCl to area | |
| :26 | 12.5 | 1.12 | :19 | 7.25 | 11.6 |
| :29 | 12.5 | 1.12 | :21 | 7.5 | 9.60 |
| :34 | 12.5 | 1.12 | :24 | 8.0 | 5.00 |
| :38 | 12.5 | 1.12 | :26 | 9.0 | 3.40 |
| | | | :28 | 10.0 | 2.01 |
| | | | :30 | 10.5 | 1.77 |
| | | | :33 | 11.0 | 1.54 |
| | | | :35 | 11.0 | 1.54 |
| | | | :36 | 11.25 | 1.42 |

To study the effect on the peripheral vagus was simple. The effect on heart rate and blood pressure of stimulating the peripheral vagus in several double vagotomized animals before and after the introduction of the drug into the pericardial sac was determined. It was found, for example, that the introduction of 2 c.c. of 5 per cent procaine into the pericardium of a 10 kilogram dog under barbital anesthesia completely

abolished the peripheral vagus effect from a moderately strong tetanic induction coil stimulus within less than three minutes. After approximately forty-five minutes a stronger stimulus showed a slight peripheral vagus effect, and after eighty minutes approximately the same peripheral vagus effect was again noted as was present before the introduction of the procaine.

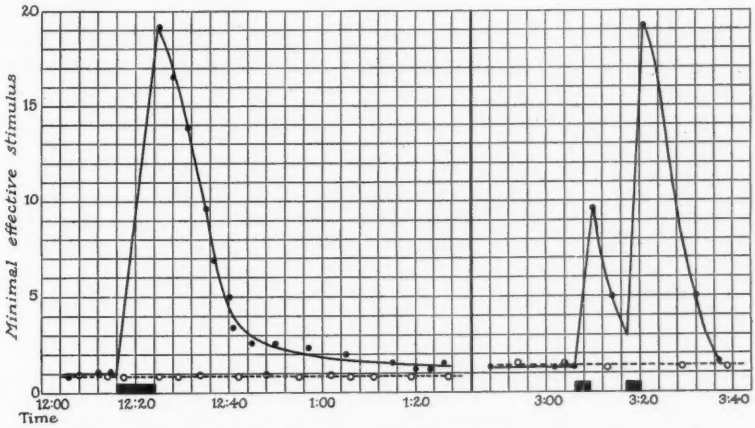


Fig. 7.—On the left are the data of Fig. 5 reconstructed on the basis of the coil calibration. On the right is an experiment in which cocaine was used. The period of diminished excitability corresponds closely with the duration of the injury component in the electrogram.

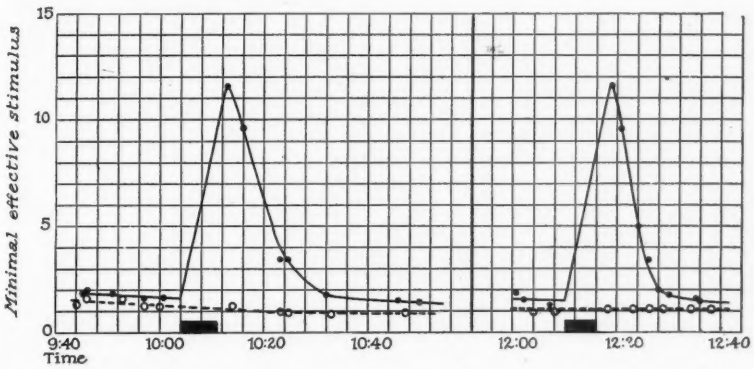


Fig. 8.—Data obtained from the use of procaine hydrochloride, left, and potassium chloride, right. Recovery is most rapid from the potassium chloride, indicating greater diffusibility.

In order to study the effect of the drugs on sinus tachycardia, we used the tachycardia produced by the introduction of a chemical irritant into the pericardial cavity. When Dakin's solution is placed on a localized area of pericardium, it produces no significant change in heart rate; but, when the entire pericardium is flooded with Dakin's

solution, a marked sinus tachycardia invariably develops. This may be due to either direct or reflex stimulation of the sinus node. As indicated graphically in Fig. 9, the development of the tachycardia can be completely prevented if an adequate amount of a local anesthetic is previously introduced into the pericardial cavity.

### OBSERVATIONS ON THE USE OF DRUGS IN EXPERIMENTAL CARDIAC OPERATIONS

Having shown that surface irritability of the heart can be decreased by local application of metycaine and procaine, I wish to discuss some observations on the use of these drugs in our experimental heart surgery. I have used these two drugs locally in preference to potassium

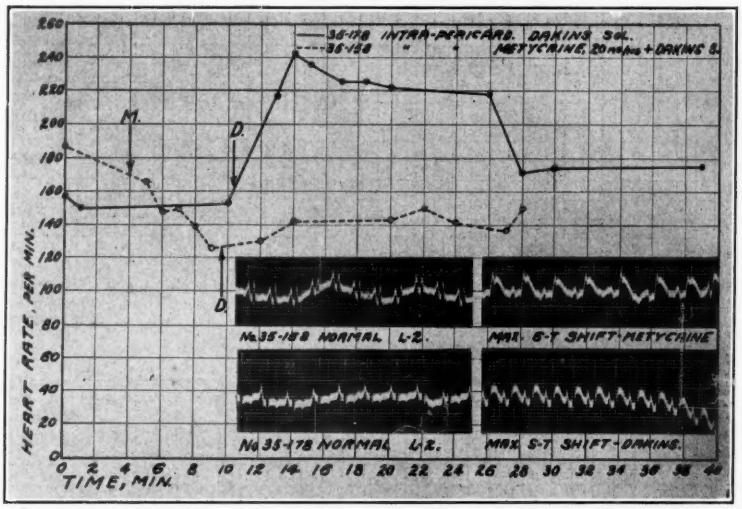


Fig. 9.—The protective action of metycaine in preventing the tachycardia due to the intrapericardial introduction of Dakin's solution. The solid line represents the pulse rate in a control, 40 c.c. of Dakin's solution being introduced into the pericardium at D. After about ten minutes the excess was aspirated and the wound closed. The dotted line represents the pulse rate in a dog following the introduction of 20 mg. of metycaine per kilogram at M, and action currents at D, the same quantity of Dakin's solution as in the other case. The epicardial absorption and myocardial action of the metycaine are indicated in the monophasic character of the electrocardiogram, Lead II.

chloride because their action is of slightly longer duration, and I believe they are less toxic in this situation.

When at operation we find a very rapid heart rate, we have been able to slow these hearts by introducing procaine or metycaine in 10 per cent solution in amounts up to 30 mg. per kilogram into the pericardial cavity, particularly over the auricles and around the base of the heart. We have introduced larger doses and have noted that the cardiac pulsations become distinctly weaker and the animal becomes cyanotic, but even in these cases recovery takes place in thirty minutes to one hour. The maximal effect of the drug develops within

less than five minutes, and by introducing small amounts cautiously until the desired effect is obtained I believe serious toxic effects can be avoided.

Occasionally, as was before mentioned, auricular fibrillation develops during an operation. In such cases the application of a small amount, approximately 1 c.c., of 10 per cent metycaine has invariably stopped the fibrillation promptly, giving a very dramatic demonstration of how a dangerous situation can be easily controlled. In these cases we were able to proceed with the operation and the animals survived.

### THE SYSTEMIC USE OF DRUGS IN CARDIAC OPERATIONS

The only drug for systemic administration adequately studied and found to be of definite value in reducing cardiac irritability in experimental cardiac surgery is quinidine sulphate. In the experimental coronary ligations many of the animals do not die because of the size of the myocardial infarct but rather as a result of increased irritability leading to the development of ventricular fibrillation. As an intermediate phenomenon we have observed ventricular tachycardias, as previously discussed.

Quinidine sulphate, in doses up to 30 mg. per kilogram given twice a day, has been found to be of definite value in controlling the ventricular tachycardias and presumably has prevented the development of ventricular fibrillation in borderline cases in which the myocardial damage was not too extensive. Figs. 10 and 11 are examples of the beneficial effect of quinidine. The observations indicate that if the myocardial damage due to coronary ligation is too extensive and severe the animals die in spite of the administration of quinidine. On the other extreme, some animals survive the same degree of coronary obstruction without the drug. The intermediate group, however, is benefited. We are at present making a statistical study of a series of right coronary artery occlusion in an effort to determine the degree of protection afforded by the administration of quinidine sulphate, and these figures will be reported at a later date.

Our observations on the use of the drug, though as yet somewhat limited, support the clinical use of quinidine sulphate in coronary thrombosis.[12, 13] The dose per kilogram corresponds to the doses reported for the control of the more difficult clinical cases of paroxysmal tachycardia.[14] We have seen no toxic effects from this dose in any of our operated animals.

Adrenalin, the classical stimulant for cardiac emergency, has been found useful but at the same time dangerous. When the heart has dilated at operation, the intracardiac injection of adrenalin has been efficacious in strengthening the cardiac contractions, but in many cases we have noted the development of ventricular fibrillation following the hypertension and hyperirritability produced by this drug. Usually when the heart dilates, the immediate emergency can be taken care of

by first removing the obvious causes of the dilatation, such as coronary obstruction or retraction, and maintaining the circulation by gentle massage. When the heart begins to recover, it will behave very much as though adrenalin had been given, showing increased irritability and hypertension during the recovery period. Our observations and those

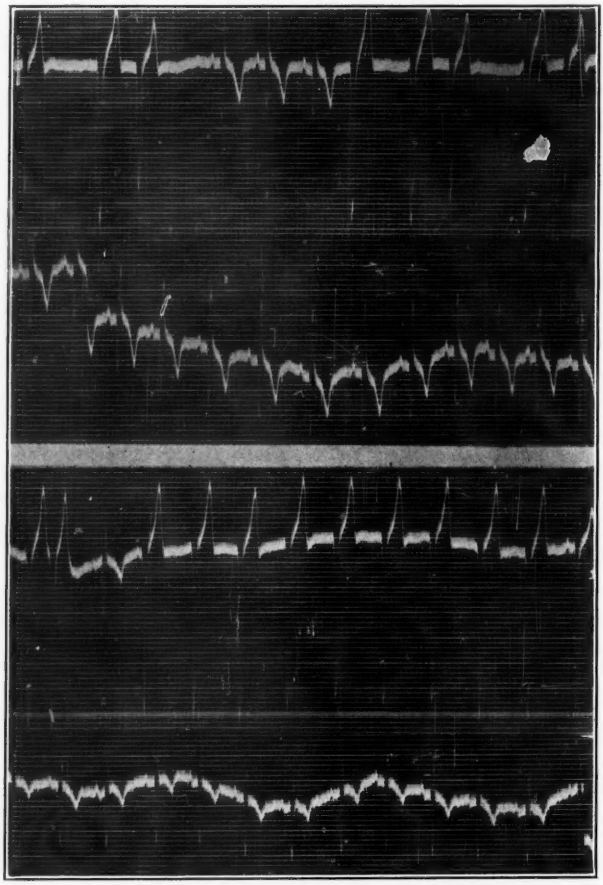

Fig. 10.—The effect of quinidine sulphate upon the ventricular tachycardia following coronary artery occlusion. Complete transformation of ventricular tachycardia into a normal mechanism by the use of 30 mg. of quinidine per kilogram as indicated in the second record taken two hours later. Twenty-four hours later the ventricular tachycardia had again reappeared and was promptly stopped with the same dose of quinidine as indicated in the third and fourth records.

of Wiggers[15] in the physiologic laboratory indicate that intracardiac adrenalin should be used only with a great deal of caution if ventricular fibrillation is to be avoided following acute cardiac dilatation.

Ventricular fibrillation should not be considered a hopeless complication, since Wiggers,[16] using the method of Hooker, Kouwenhoven

and Langworthy, was able to revive 80 per cent of the hearts that had been fibrillating five to seven minutes in the physiologic laboratory. His modification of the technic has been first to massage the heart gently in order to fill the coronary vessels with oxygenated blood, then allow a brief passage of 60 cycle alternating current with a minimal strength of 1 ampere to pass between padded electrodes placed in contact with either side of the heart. Using this method, we have been able to restore normal rhythm after the onset of ventricular fibrillation in the surgical laboratory, and I am convinced that every

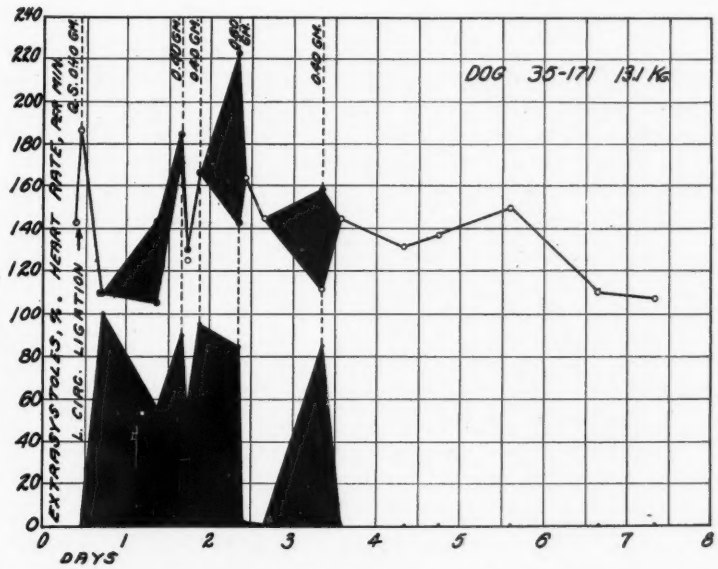


Fig. 11.—A ventricular tachycardia following the production of an infarct in the left circumflex region. Heart block developed several hours after the coronary occlusion. On the following day a ventricular tachycardia of variable rate was present, and the rate continued to increase until quinidine was given, following which there was an abrupt drop in rate and in the percentage of extrasystoles. The effect of the third dose of quinidine is not evident because no observations were made during the night. The following day a terrific tachycardia was present and was immediately controlled with quinidine. A further reappearance of the tachycardia was completely controlled by quinidine.

cardiac operating room should be equipped with such an apparatus so that the electrodes can be applied in a moment's notice should ventricular fibrillation develop.

## SUGGESTIONS FOR THE APPLICATION OF THESE DATA TO HUMAN SUBJECTS OF CARDIAC OPERATIONS

On the basis of the observations reported in this paper it is believed that the use of drugs, both on the epicardium and systemically, in human subjects of cardiac operations will prove of value. If, for example, quinidine sulphate were given preoperatively, it might well

prevent the development of auricular and ventricular fibrillation during the operation or postoperative period. On the other hand, if at operation a heart appeared extremely irritable or if a tachycardia or auricular fibrillation developed, these disturbances could be controlled by the cautious intrapericardial administration of procaine. Without a doubt excessive amounts of these drugs are toxic, and careful observations and experience will be necessary to determine their ultimate value in human cases. No one would deny that adrenalin belongs in every emergency drug set-up, and yet hearts have been thrown into ventricular fibrillation and all chance of recovery destroyed by the indiscriminate use of this drug.

I wish to express my appreciation to Dr. Claude S. Beck for his help in planning this study; also to Dr. C. J. Wiggers and Dr. Torald Sollmann for valuable suggestions and criticisms; and to Dr. Carl A. Hamann, Jr., for assisting with a number of the experiments.

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# A STUDY OF CARDIOPERICARDIAL ADHESIONS*

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THE generally accepted conclusion that pericardial adhesions play a fundamental rôle in the production of cardiac hypertrophy has been widely taught and approved. A few selected sentences from standard textbooks illustrate the foregoing statement: "If the adhesions be extensive a hypertrophy of the heart occurs varying in degree with the extent of the adhesion and the condition of the surrounding regions. . . . Certain authorities insist that there must be external adhesions, but we have rarely failed to see hypertrophy in extensive pericardial adhesions regardless of external conditions." (Karsner.[1]) "Adhesions not only between the epicardium and pericardium, but between the pericardium and pleura, or chest wall cause the heart to work under considerable difficulties and are therefore one of the most certain causes of cardiac hypertrophy." (Beattie and Dickson.[2]) "The influence of pericardial adhesions in causing cardiac hypertrophy through increase of the work to be performed is of course well known." (Krumbhaar.[3]) "There are two forms of adherent pericardium. . . (b) Adherent pericardium with chronic mediastinitis and union of the outer layer of the pericardium to the pleura and chest wall. This constitutes a most serious form of cardiac disease, particularly in early life, and may lead to an extreme grade of hypertrophy and dilatation." (Osler.[4]) Referring to cardiac enlargement, White[5] in 1931 stated: "It is not present unless extra work has been required of the heart because of adhesion to chest wall, diaphragm, or mediastinum. When the handicap has been marked the heart may be very much enlarged. . ." Apparently Hope,[6] in 1832, was the first to propose that pericardial adhesions caused cardiac hypertrophy, and he remained its leading exponent for many years. Although the definite factors concerned in the production of cardiac hypertrophy in the presence of cardiopericardial adhesions are not well established or known, no experimental confirmation of note was forthcoming until Herrmann, Musser and Ochsner[7] supported Hope's teaching by their work on experimental pericarditis. They found that "in dogs with partial synechia of the

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Read (by invitation) at the Nineteenth Annual Meeting of the American Association for Thoracic Surgery at Rochester, Minnesota, May 4, 5, 6, 1936.

This investigation was aided by a grant from the Josiah Macy, Jr. Foundation.

pericardium associated with anterior mediastinitis, there was a distinct cardiac hypertrophy with a definite increase in the preponderance of the right ventricular wall in all those that had had the lesions more than 80 days. . . The pathologic changes were conspicuous in all dogs in which the lesion had been present over 30 days, and in one dog in which the lesion had existed only 17 days.'' They concluded that intrapericardial, as well as extrapericardial, adhesions are significant and produce cardiac embarrassment.

In contrast to the previously mentioned statements pertaining to pericardial adhesions are the following statements which have been more or less unaccepted and are infrequently found in the literature. In 1842 Chevers[8] refuted Hope's opinion that pericardial adhesions lead to enlargement of the heart, showing that they have rather the contrary effects—obliteration of the cavity tends to cause diminution of the heart and great vessels. He reported that from 1835 to 1842 at Guy's Hospital there had been no case of hypertrophy occurring in combination with obliteration of the pericardial cavity which failed to show valvular disease or other cause for the enlargement. Stokes[9] and Laennec[10] made similar statements. In 1845 King[11] wrote a paper entitled ''The Harmlessness of Adherent Pericardium.'' These pioneers, however, did not have a clear-cut conception of pericardial adhesions, nor did they separate the subject into its individual components. Tice[12] states: ''The manifestions of adhesive pericarditis are those of a chronic heart disease, not due primarily to the adhesions, but to the associated cardiac pathology.''

Further doubt was cast upon the established point of view by many experiments carried out during the past twelve years by Beck[13] and his associates on problems relating to the surgery of the heart and pericardium. With these observations in mind, this problem concerning adhesions to the heart was to be put to a critical experimental test. An additional reason for the study of this problem—a practical reason—is the fact that the myocardium can be given a supplementary blood supply through tissues grafted upon the heart. The Beck operation[14] involves myocardial adhesions, and the rôle of adhesions needs critical study if coronary sclerosis is to be treated by this new surgical principle. The study of this problem could also be applied to the Brauer operation, in relationship to pericardial adhesions.

## EXPERIMENTAL METHOD

During the past two and one-half years we have directed our attention to the production and consideration of intrapericardial and extrapericardial adhesions associated with moderate displacements of the heart. The methods employed were routine and as follows: Healthy dogs, weighing about 19 kg., were used. Control studies consisted of weight, venous pressure, anteroposterior roentgenograms of the chest,

and electrocardiograms taken in the three standard positions. Following this the dogs were indiscriminately divided into two groups. The first group consisted of thirteen dogs. In seven the parietal pericardium was sutured to the dome of the left diaphragm, Fig. 1. As much of the pericardium was sutured as was anatomically and technically possible. In the remaining six dogs a similar procedure was carried out on the right side. Here the parietal pericardium was sutured to the dome of

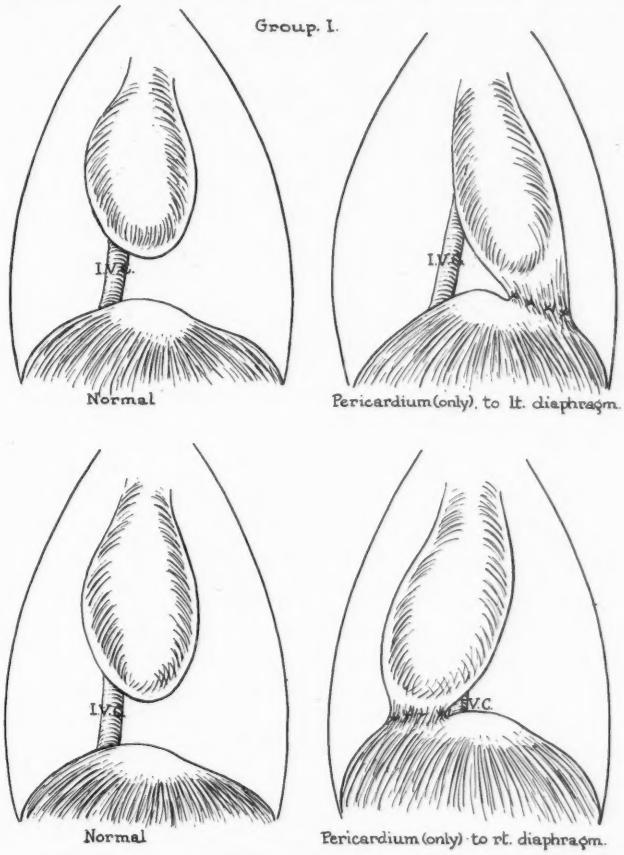


Fig. 1.—Diagram showing relationship of heart to diaphragm in the animals before operation, and after the pericardium had been attached to either the left or the right diaphragm. In the latter the contour of the heart and diaphragm are obviously altered.

the right diaphragm anterior and lateral to the inferior vena cava. The heart was not included in these sutures. It was entirely free within the pericardial sac.

Another group was composed of thirteen dogs. It was our purpose in this group to anchor the ventricles to the diaphragm. This proce-

dure was carried out in two stages. At the first operation parietal pericardium was attached to the ventricle by superficial sutures, Fig. 2. Six weeks were given for these structures to grow together. Control studies were then repeated and found to be normal in all respects. The parietal pericardium was then sutured to either diaphragm as was done in the first group of experiments. In this second group, however, the

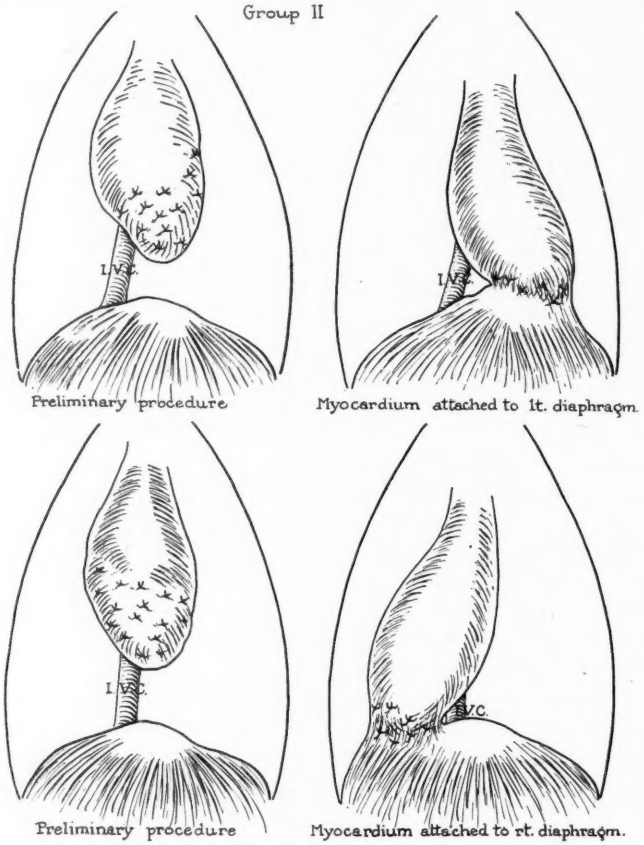


Fig. 2.—In this group the pericardium was first attached to the ventricle by superficial sutures. Following control studies the pericardium and attached myocardium were sutured to either diaphragm.

heart, pericardium, and diaphragm were attached; whereas in the first group parietal pericardium and diaphragm alone were attached. The preliminary operation was carried out in the second group in order that at a second operation it would not be necessary to take deep sutures into the underlying myocardium in order to anchor it to the domes of the right and left diaphragms respectively. During these operations it was

at times difficult to tie the sutures because of the tension which developed upon downward movement of the diaphragm. With the heart anchored to the right side, a moderate constriction of the vena cava was sometimes noticeable on inspiration. This slight constriction of the inferior vena cava was of no physiologic importance.[15]

The animals were observed and studied frequently. Six dogs in which good adhesions were present were selected for strenuous exercise upon a treadmill. At the termination of the experiment the animals were killed with ether, and autopsy examination included examination of the thoracic and abdominal organs. Microscopic sections of the ventricles were made, and the thickness of the ventricles was recorded.

## CLINICAL OBSERVATIONS

The animals recovered rapidly from the operative procedures. A moderate number of them shortly after operation displayed diaphragmatic irritation as evidenced by hiccough-like respiration; however this affection cleared up in the course of a few days. Broadbent's sign was demonstrable in ten dogs. It was seen in seven in which the heart was displaced to the right. In five of these the ventricle had been anchored to the diaphragm; whereas in two only the pericardium was attached. Only three demonstrated this sign with the heart displaced to the left. In all of these the heart, rather than the pericardium alone, was attached to the left dome of the diaphragm.

Not a single instance of circulatory embarrassment was encountered. The venous pressures remained normal in each experiment. Tolerance for vigorous exercise on a treadmill was normal. Roentgenograms of the chest showed a change in the general conformation of the heart shadows—changing grossly from a globular shadow to an elongated one, Fig. 3. Various degrees of displacement were noted. The width of the cardiac silhouette was not necessarily indicative of the size of the heart, because of its obvious change in position and possible rotation. Fluoroscopic examination indicated that with descent of the diaphragm a downward pull was exerted upon the pericardial sac causing the sides of the sac to become taut, the heart to elongate, and possibly tending to constrict it at its widest portion. At the site of the adhesions the diaphragm was pulled up. The electrocardiographic findings were for the most part inconsistent and inconclusive. It will suffice to say that after adhesions were created, in the majority of the records the electrical axes were stationary with change in position. This was especially true in those groups in which intrapericardial adhesions were present.

Before the animals were killed, the chest was opened under ether anesthesia, and the movements of the heart and diaphragm were observed. Often pleural adhesions had to be freed before a view of the heart and pericardium could be obtained. It was readily seen that with descent of the diaphragm the heart was definitely pulled down.

This downward pull upon the pericardium or heart produced an elongation and a narrowing of the heart. The effect of this pull was also noticeable upon the pulmonary veins and superior vena cava. These vessels, however, were not actually kinked by the downward pull. The greatest pull upon the heart occurred when both intrapericardial and extrapericardial adhesions were present. In spite of the fact that similar areas for the adhesions were selected in all cases, the greatest

Fig. 3 *A*. Fig. 3 *B*.

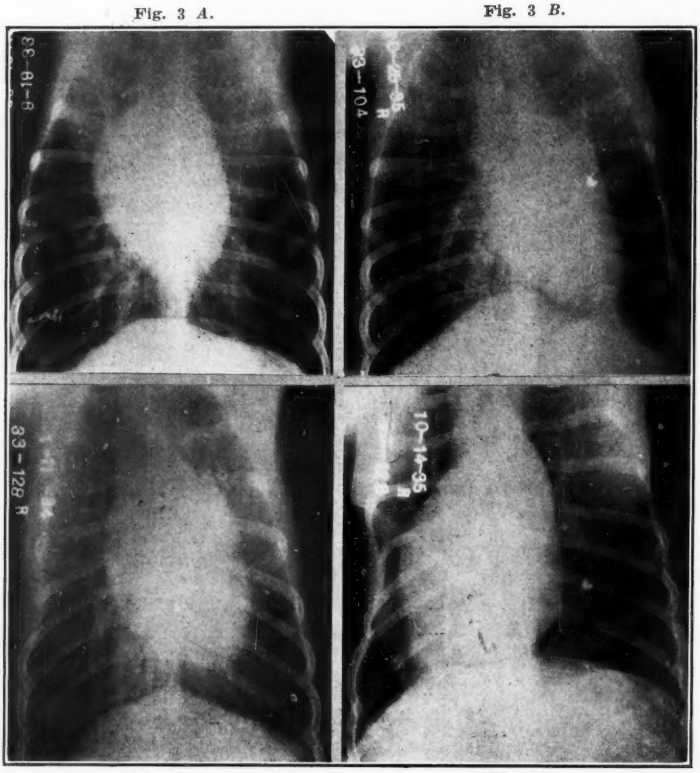

Fig. 4 *A*. Fig. 4 *B*.

Fig. 3.—*A*, X-ray of heart before pericardium was attached to the diaphragm. *B*, X-ray taken in full expiration. Heart is elongated and displaced to the left. The space between the heart and the diaphragm is almost obliterated, and the diaphragm has a double contour.

Fig. 4.—*A*, X-ray taken after preliminary operation. *B*, Myocardium, pericardium, and diaphragm adherent. Heart definitely displaced into right chest with change of contour of diaphragm and heart.

displacement generally occurred when the heart, rather than the pericardium alone, was adherent to the diaphragm, Figs. 4 and 5. Less displacement of the heart occurred in those dogs in which only the pericardium was attached to the diaphragm. The adherent portion of pericardium was folded and wrinkled at the site of the adhesion to the

diaphragm in this latter group. In other words, the parietal pericardium had stretched and accommodated itself.

## NECROPSY

Pleural adhesions of varying extent and firmness were encountered. In many instances the lower lobes of the lungs were adherent to the lateral pericardium, diaphragm, chest wall, and pleural sinus. In one animal the pleural sinus and almost the entire pleural cavity were obliterated, Fig. 6. This was caused by a sponge that was inadvert-

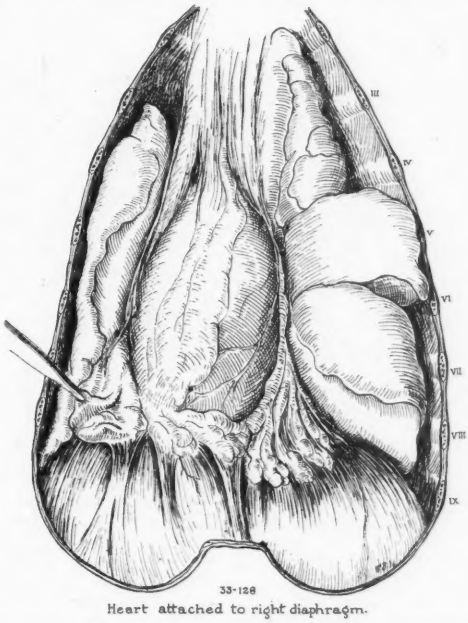


Fig. 5.—Same animal as in Fig. 4. The heart is moderately displaced to the right. The right lower lobe of the lung is adherent to both pericardium and diaphragm.

ently left in the chest. The area of the heart adherent to the diaphragm usually measured about 25 sq. cm.

The observation of greatest importance was that cardiac hypertrophy was not found in any of these experiments either grossly or microscopically.* In not a single instance did we find hypertrophy of the heart. Herrmann[16] established a mean ratio between heart weight and body weight for normal dogs (0.00798). After carefully weighing our animals and their dissected hearts we were unable to find any significant change from the normal ratio of heart weight to body weight. In other words, the mean ratio was less than normal but within normal limits. The mean ratio for that group in which the heart was displaced to

*We are indebted to Dr. M. A. Simon for his study of the microscopic specimens.

the left by the attachment of the pericardium alone was 0.00713; while the ratio for a similar group to the right was 0.00696. With combined adhesions and displacement to the left the mean ratio was 0.00704, and was 0.00724 with combined adhesions and displacement to the right.

In the experiments in which the right ventricle was adherent to the right diaphragm a definite sacculation or aneurysm of the right ventricular wall was noted, Fig. 7. The myocardium in this region was of normal thickness. This sacculation was due to the pull exerted by the diaphragm, Fig. 8.

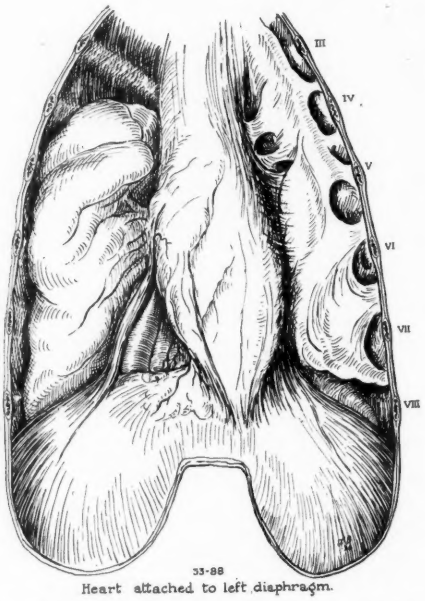


Fig. 6.—Combined adhesions with slight displacement of the heart. Practically the entire left pleural cavity and the left pleural sinus were obliterated in this animal.

Numerous other experiments dealing with the production of pericardial adhesions of varying extent and location were carried out. No instances of cardiac hypertrophy have been disclosed. Circulatory embarrassment occurred only in those in which chronic cardiac compression took place.[17] One animal was operated upon in March, 1932, and the pericardium was attached directly to the chest wall. Later, Dakin's solution was injected intrapericardially. The animal developed signs of chronic cardiac compression. Resection of a window of the anterior pericardium was carried out, and at the same time the heart was sutured to the diaphragm. The venous pressure quickly returned to normal and the dog remained healthy. The animal was killed in November,

1935. Extensive pericardial adhesions were encountered, while no evidence of cardiac hypertrophy was found, Fig. 9.

## COMMENT

Pericarditis in the human being is not caused by mechanical factors or chemical irritation; in reality, it usually results from a pancarditis

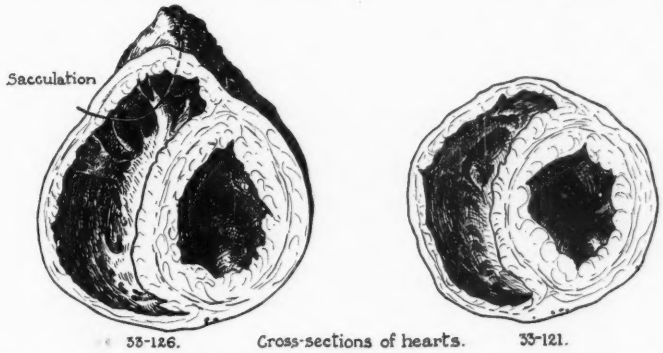


Fig. 7.—Cross-sections of two hearts taken at similar levels. In No. 33-126 a small sacculation of the right ventricular wall was noted in the region which corresponded with the center of the adhesion. In No. 33-121 the myocardium was not included in the sutures. No hypertrophy is noted in either heart.

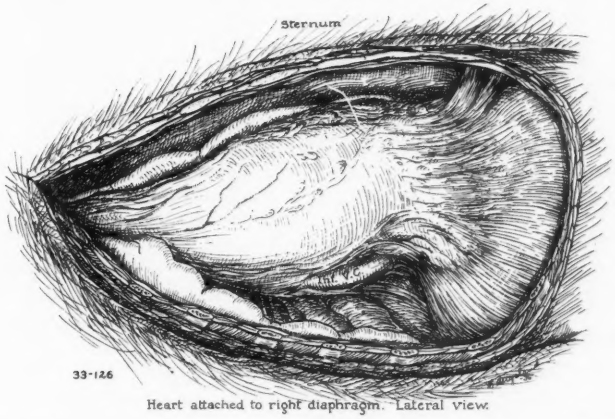


Fig. 8.—Drawing of Experiment 33-126. Lateral view of extensive adhesion.

or not infrequently from advanced tuberculosis. These conditions result from infections which progress from an acute stage into one of chronicity, and they are not necessarily manifest only in the pericardium.

In attempting to produce cardiac hypertrophy in dogs one should be able in this period of time to demonstrate at least a degree of hyper-

trophy which should be significant. Inasmuch as we could not, it lends some doubt to the previous teaching and suggests that some other influence has been at work.

As paradoxical as it may seem, we are led to believe that adhesions per se do not cause circulatory embarrassment unless they are extensive enough to cause cardiac compression; we believe that they have been overemphasized in the past.[18] We are supported in this assumption by a study of the autopsy records of the University Hospitals of Cleveland for the period from 1906 to 1935. In 4,400 autopsies there

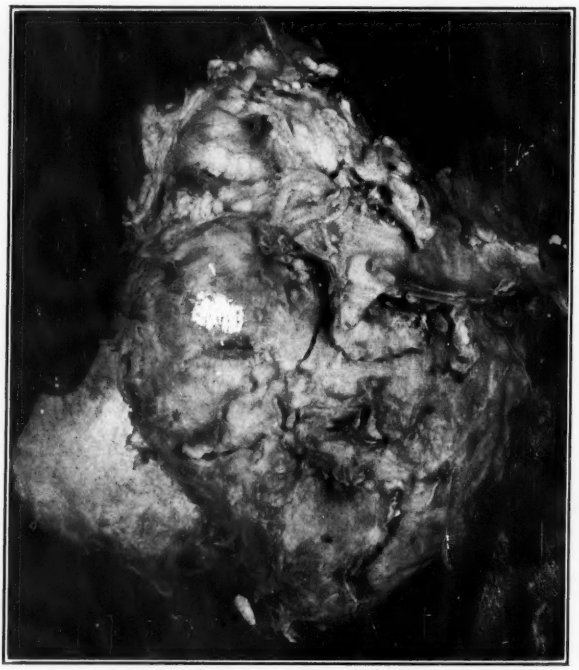

Fig. 9.—Dog 32-55, weight 20.1 kg., heart weight 140 grams. Generalized adhesions between chest wall, lungs, mediastinum, and diaphragm. Duration of experiment was three years and eight months.

are 76 cases in which extensive pericardial adhesions were found, Table I. Only those cases were included in which the adhesions were extensive and combined with intrapericardial adhesions. Seldom had the diagnosis been made clinically. In 54 cases in which the hearts had undergone hypertrophy there was concomitant heart disease or vascular disease which in itself could account for the hypertrophy; for instance, rheumatic pancarditis, aortic insufficiency, myocarditis, etc. (agreeing with Chevers'[8] unaccepted observations in 1842). There was not a single instance in which an adequate explanation was not found. In

21 instances extensive pericardial adhesions were found associated with hearts of either normal or smaller than normal size which were entirely free from valvular or vascular disease. Almost without exception the largest hearts were the seat of rheumatic pancarditis.

TABLE I

AUTOPSY MATERIAL, UNIVERSITY HOSPITALS, CLEVELAND, OHIO, 1906-1935

| | |
|---|---|
| Total number of cases of extensive cardiopericardial adhesions<br>Number of autopsy records 4,400 Incidence 1.7% | 75 |
| A. Cases associated with cardiac hypertrophy | 54 |
| a. In all these cases there was concomitant heart disease or vascular disease which in itself could account for the hypertrophy | |
| b. Almost without exception the largest hearts were the seat of rheumatic pancarditis | |
| B. Cases without cardiac hypertrophy | 21 |
| a. These hearts were free from valvular, myocardial, or vascular disease in all except one case and this showed mild rheumatic heart disease<br>(Agreeing with Chevers' report of 1842) | |

Some might contend that the combination of the cardiac disease and the pericardial adhesions led to the production of a larger heart. With this autopsy material in mind, the burden of proof rests on the one who makes such a contention.

The hearts encountered in the chronic cardiac compression syndrome (Pick's disease) are small, regardless of the fact that extensive intrapericardial and sometimes extrapericardial adhesions are found.[17] Extensive pleuropericardial adhesions and mediastinitis are found also in advanced cases of pulmonary tuberculosis, and it is exceptional to find the heart in its normal position; yet, in spite of these conditions being present for a number of years, the heart is usually small, and symptoms referable to the circulatory system are absent.[19, 20] To recapitulate, we are led to believe that adhesions per se do not cause circulatory embarrassment unless they are extensive enough to cause cardiac compression, angulation or torsion. In view of our experiments and analysis of autopsy material, we believe that pericardial adhesions in general, and to the diaphragm in particular, do not play a significant rôle in the production of cardiac hypertrophy. The beneficial results of and the indications for the Brauer operation have probably been overemphasized in the past.

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# SUPERIOR VENA CAVAL THROMBOSIS

## Review of the Literature and Report of Cases of Traumatic and Infectious Origin

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OBSTRUCTIONS to the superior vena cava whether partial or complete are rare clinical and pathologic entities. Ehrlich, Ballon, and Graham[1] in 1933 were able to collect from the world literature only 309 cases. Obstructions due to thrombosis occur much less frequently than other types, and superior vena caval thrombosis is the rarest of all venous thromboses. We have been able to collect in an exhaustive review of the world literature only 120 cases of superior vena caval thrombosis, all of which, however, were not definitely proved. In the present report, which is based on an analysis of the 120 reported and 2 unreported cases, only thrombosis of the superior vena cava will be considered.

*Historical.*—According to Service,[2] Schenck,[3] in 1644, was the first to describe obstruction of the vena cava. An analysis of Schenck's original paper, which was written in Latin, reveals, however, that the only reference in it to veins concerns bloodletting in dysentery, which was done because of the belief that bile, supposedly responsible for the dysentery, was removed in the blood. According to Zambellini[4] the first two reported cases of superior vena caval thrombosis were by Bartolino[5] and Hunter.[6] In 1808, Marjolin[7] reported a case of superior vena caval obstruction by a clot extending from the opening in the auricle to the trunk of the left subclavian vein. Deckart,[8] in 1823, Otto[9] and Bouillaud,[10] in 1824, Weissbrod[11] in 1831, Ried[12] in 1835, and Martin-Solon[13] in 1836, each reported a case. Hardwicke[14] in 1845 reported two cases. Stannius[15] in 1839, Paulus[16] in 1842, Bouchut[17] in 1845, Watson[18] in 1847, and Turnbull[19] and Barth[20] in 1853, reported isolated instances; and Duchek[21] in 1854 reported 5 cases of superior vena caval thrombosis. In addition to these, we have been able to collect from the literature 98 cases which have been reported since 1854, making a total of 120 cases.

*Etiology.*—According to Fischer[22] the causes of superior vena caval obstruction are as follows: complicating thrombus from the periphery, 4 per cent; localized phlebitis with thrombus formation, 1.5 per cent;

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tuberculous plebitis, 1.5 per cent; actinomycosis, 0.5 per cent; contraction or compression by scar tissue, 6 per cent; obliteration of the pericardium with narrowing of the superior vena cava by cardiac and respiratory movements, 0.5 per cent; primary thoracic tumors, benign 4 per cent, malignant 37 per cent; extrathoracic malignant tumors, metastatic, 4.25 per cent; tumors from invasion of the superior vena cava, 4.25 per cent; metastasis into the vein wall but not into the lumen, 0.5 per cent; aneurysm, 36 per cent. In this group were included superior vena caval obstructions not due to thrombosis. Brown,[23] in 300 reported cases, found that aneurysm of the aorta was the cause in 55 per cent, carcinoma of the lung, bronchi, and adjacent organs, 25 per

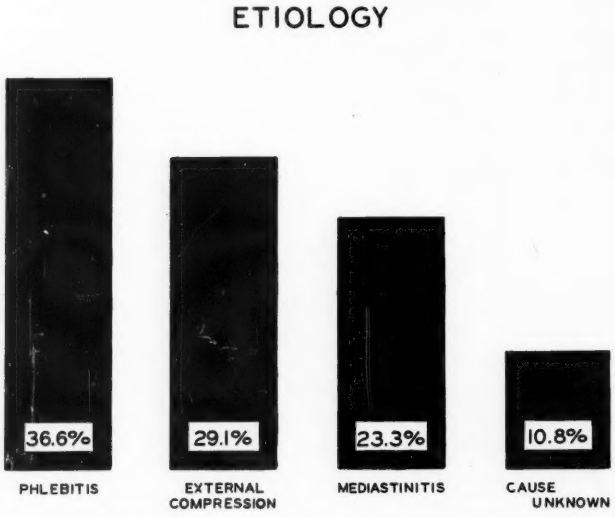


Chart 1.—Chart showing comparative instances of etiologic factors.

cent, and in the remaining 20 per cent there were various etiologic factors. In contradistinction to the above mentioned reports the 120 cases analyzed in the present communication are limited to thrombosis of the superior vena cava and do not include obstructions due to other causes.

Thrombosis of the superior vena cava is the result of injury of the vena cava by some agent, which may be either in the wall of the vein or as a result of pressure from without. We have classified the collected cases according to their etiology. Forty-four (36.6 per cent) resulted from phlebitis, 35 (29.1 per cent) from external compression, 28 (23.3 per cent) from mediastinitis, and in 13 (10.8 per cent) the cause either was not stated or was unknown (Chart 1). Of the 44 cases of phlebitis in which there was an associated thrombosis of the superior vena cava, 20 (16.6 per cent) were classified as idiopathic (10, 17, 24-

31, 32 [Case 1], 33, 34 [Cases 1, 2, 3], 35-39). Of this group there were 10 in which there was an associated heart disease (10, 17, 29, 30, 31, 34 [Cases 1, 2, 3], 37, 39). There were 12 (10 per cent) cases of

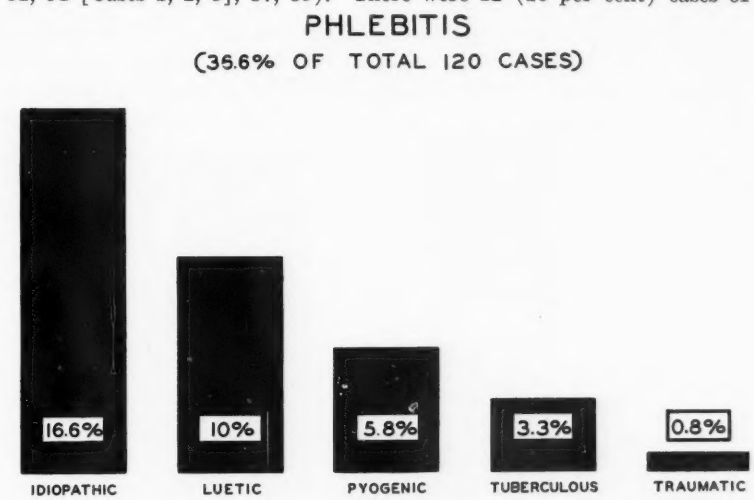


Chart 2.—Graphic chart showing comparative instances of various types of phlebitis resulting in superior vena caval obstruction.

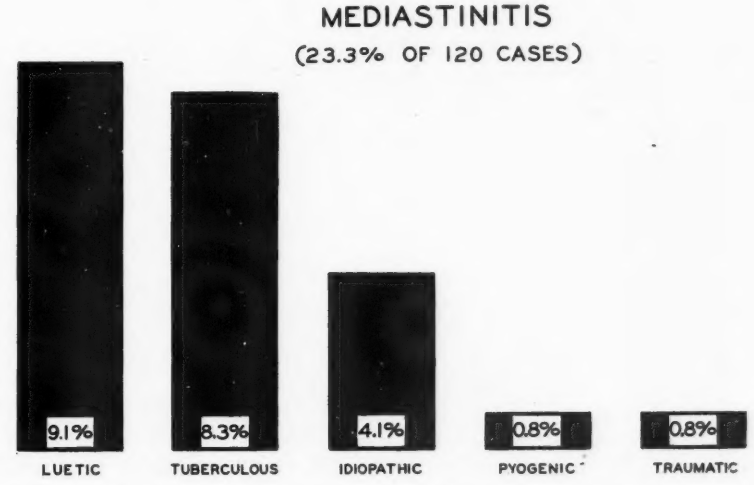


Chart 3.—Graphic chart showing comparative instances of causes of mediastinitis resulting in superior vena caval thrombosis.

luetic phlebitis (4, 40, 41 [Cases 1, 2, 3], 42, 43 [Cases 1, 2], 44-46, 105); 4 (3.3 per cent) cases with tuberculous phlebitis (47-50); 7 (5.8 per cent) cases of pyogenic phlebitis (16, 21, 51-55), and 1 (0.8 per cent) case of traumatic phlebitis (56) (Chart 2).

Of 28 cases in which the cause was an antecedent mediastinitis, there were 5 (4.1 per cent) in which the etiology was not known, and there-

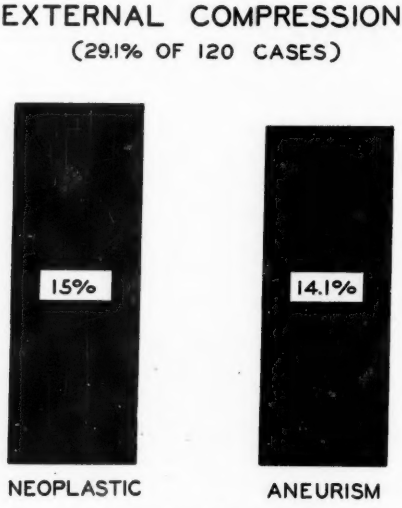


Chart 4.—Graphic chart showing comparative instances of external compression producing superior vena caval thrombosis.

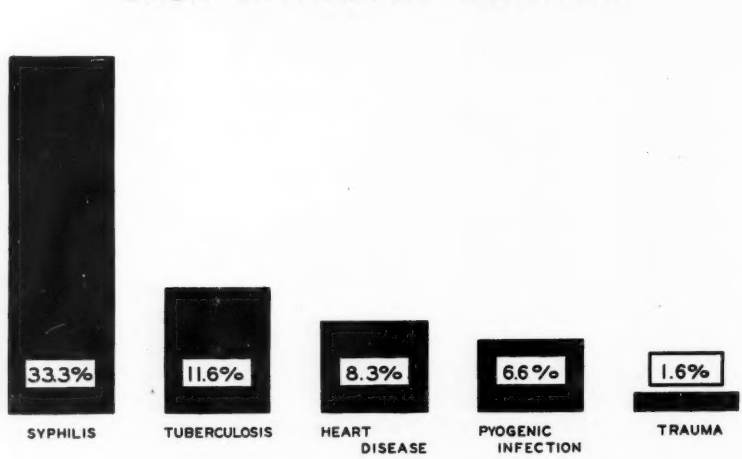


Chart 5.—Graphic chart showing comparative frequencies of basic pathologic conditions.

fore considered idiopathic (12, 57-60). Lues was responsible in 11 (9.1 per cent) (21, 41, 43, 61-67 [Cases 1, 2]); tuberculosis in 10 (8.3 per

cent) (21 [Case 5], 50 [Case 2], 68-70 [Case 2], 71-74 [Case 1], 75); pyogenic infections in 1 (0.8 per cent) (76), and trauma in 1 (0.8 per cent) (77) (Chart 3).

There were 35 cases of superior vena caval thrombosis caused by external compression. Seventeen of these were caused by aneurysm (6, 8, 9, 13, 16, 18, 21 [Case 3], 78-83 [2 cases], 84, 86 [2 cases]), and 18 by neoplasm (11, 15, 19, 20, 23, 73, 74 [2 cases], 87-96) (Chart 4). Cases in which the vena caval obstruction was the result of the filling of the lumen with tumor tissue are not included in the present report. There were 13 cases in which the cause of the thrombosis was not stated or the cause was unknown (5, 7, 97-101 [4 cases], 102-104).

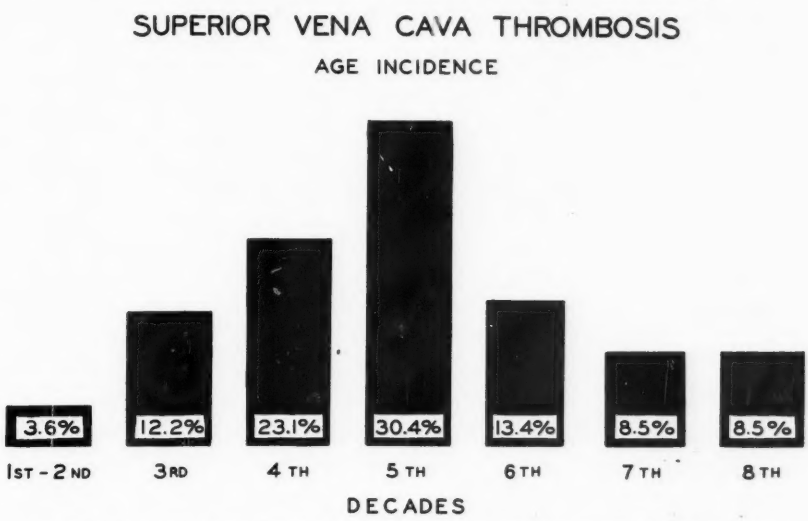


Chart 6.—Graphic chart showing age incidences in reported cases of superior vena caval thrombosis.

Syphilis was responsible either directly or indirectly in 40 (33.3 per cent) of the 120 cases; 12 had luetic phlebitis, 11 luetic mediastinitis, and 17 aneurysm. Tuberculosis caused the thrombosis in 14 (11.6 per cent) with tuberculous mediastinitis in 10 and phlebitis in 4. There was an associated heart disease in 10 (8.3 per cent). Pyogenic infections were responsible in 8 (6.6 per cent), 7 having phlebitis and 1 mediastinitis. Trauma was an etiologic factor in only 2 cases (1.6 per cent), 1 with mediastinitis and the other with phlebitis (Chart 5).

*Incidence.*—In the 120 cases statements were made concerning the sex in 95. There were 64 males (67.2 per cent) and 31 (32.6 per cent) females. In the 95 cases there were only 3 colored patients (3.1 per cent). A statement concerning the age was made in 82 instances; the oldest was 74 years, the youngest 9 years, and the average 43.6 years.

Fifty-three per cent of cases occurred in the fourth and fifth decades with the highest incidence (30 per cent) in the fifth decade. Only 3.6 per cent occurred in the first two decades, 12.2 per cent in the third decade, 13.4 per cent in the sixth decade, and 8.5 per cent each in the seventh and eighth decades (Chart 6).

*Clinical Manifestations.*—Clinical manifestations of superior vena caval obstruction are dependent upon stasis of blood in the tributaries draining into this vessel and, therefore, are limited largely, if not wholly, to the upper half of the body. Because of the obstruction in the superior vena cava and because of the necessity of short-circuiting the return flow of blood from the head, the neck, and the upper extremities into the branches of the inferior vena cava, stasis results, causing increased venous pressure in this portion of the venous system in contrast to the normal venous pressure in the remaining portions of the body. According to Lian and Abaza[106] elevated venous pressure in the upper half of the body with normal venous pressure in the lower portions is indicative of superior vena caval obstruction; whereas elevated venous pressures in both the upper and the lower portions of the body are found in cardiac failure. Whereas there is considerable variation in the normal venous pressure, pressures over 15 cm. of saline obtained with the patient in the horizontal position under basal conditions are abnormal. Carlson[107] in experimental superior vena caval ligations found that immediately after ligation above the azygos the venous pressure rose 100 per cent; at the end of nine days it was 65 per cent above normal, and in fourteen to twenty-nine days it was not significantly increased. Following obstruction of the superior vena cava and azygos the venous pressure seven days after the second procedure (azygos ligation) was increased 167 per cent. Thirty-six days after the second stage the pressure in the jugular vein was 10 cm. and that in the femoral was 5 cm. of saline.

Because of venous stasis and deficient drainage, transudation of fluid outside the vessel occurs with resultant edema and swelling of the affected area. The effect of increased venous pressure on transudation of fluid is shown by the investigations of Landis, Jonas, and Angevine.[108] Krogh, Landis, and Turner[109] demonstrated that a filtration of fluid into the tissue occurs when venous pressures are greater than 15 to 20 cm. of water and that above 17 cm. the rate of filtration is directly proportionate to the increase in venous pressure. This increase in transudation of plasma in the presence of increased venous pressure is possibly the result of two factors, increased resistance and anoxemia, which accompany obstruction to the superior vena cava. Landis[110] has shown that anoxemia increases the permeability of the vessels with resultant edema. The edema of the upper half of the body with superior vena caval obstruction is frequently not severe enough to cause pitting. This is

probably due to the fact that collateral veins develop sufficiently to compensate for the venous obstruction and also because of the nondependent position of the upper half of the body. The edema and swelling of the face, neck, and upper extremities are dependent largely upon the posture; it becomes less when the patient is up and about and in the upright position, and almost invariably is aggravated when the patient is recumbent. Edema of the eyelids is frequently an early manifestation. According to Rauth,[112] edema of the glottis may be so severe as to cause death.

Cyanosis of the upper half of the body is another prominent manifestation in patients with superior vena caval thrombosis. Rather characteristically the cyanosis is limited to the face, being most prominent in the lips and the ear lobes. At times the cyanosis is so marked that a generalized lividity of the face is present. Cyanosis of the finger tips is not marked, although it can occur in extreme cases. Cyanosis probably is largely due to anoxemia resulting from stagnation of blood in the tributaries of the superior vena cava. According to Ernstene and Volk,[111] however, venous stasis does not alter the rate of exchange of carbon dioxide and oxygen between the blood and the skin. Osler[73] believes that cyanosis is due to incomplete oxygenization of the blood in the lung because of the dilatation and thickening of the collateral between the bronchial and the pulmonary vessels. This results in insufficient or incomplete gaseous exchange between the air in the alveoli and the blood in the pulmonary capillaries. According to Rauth,[112] cyanosis is the result of translucency of the markedly distended cutaneous vessels. We are of the opinion that anoxemia is largely responsible for the development of cyanosis, because it is markedly aggravated when stasis is increased, particularly when the patient assumes the horizontal position. This was well demonstrated in our Case 1, in which cyanosis greatly deepened when the patient was recumbent and decreased considerably when the patient was in the upright position.

Dyspnea is a frequent manifestation, but its cause is not well understood. According to Arrillaga and Taquini,[113] as a result of superior vena caval compression there is a slowing of the local circulation which results in abnormal accumulation of carbon dioxide in the blood. This produces a great arteriovenous oxygen difference. Because of the poor low content of the oxygen and rich content of the carbon dioxide, there is hyperventilation, which according to these authors is due to bathing of the respiratory centers in blood, rich in carbon dioxide. Dyspnea is also frequently aggravated when the patient is in the horizontal position. Waterfield[58] reports a case in which there was Biot type of respiration when the patient was asleep. This patient when sitting up had relatively little respiratory difficulty. At times when the patient was in lighter sleep, breathing was of the Cheyne-Stokes type.

Pleural effusion may occur as a result of the compression of the vena azygos major. In such instances, according to Bettman,[114] the pleural effusion is usually on the right, but is later followed by a similar effusion on the left. This may also be associated with an edema of the chest wall. Pericardial and mediastinal effusion may occur according to that author.[114] In a case reported by Wilkins[56] a bilateral hydrothorax developed, which was most marked on the left, necessitating aspiration from June 27 to October 13 of 862 ounces of fluid. In an analysis of the reported cases it was found that unilateral hydrothorax was always on the right side and in but one instance in a case with bilateral involvement was the greatest amount on the left (Wilkins[56]).

Cough is frequently an early manifestation. When present, it is probably due to the underlying lesion which is responsible for the thrombosis of the superior vena cava or a result of the stasis. In the former instance, it may be due to a tuberculous lesion of the lung or mediastinum. In the latter instance, the edema of the mediastinum with compression of the pulmonary vessels and bronchi, producing stasis in the lung, may be the important factor. Cough may result from irritation of the upper respiratory tract, especially the larynx, in those cases in which there is edema and congestion of this organ. Hemoptysis occurs infrequently, and, when present, it is probably due to underlying lesions in the lung, such as a tuberculous process or mediastinal lesions which exert pressure on the pulmonary vessels producing stasis in the lung parenchyma.

As a result of stasis in the cerebral vessels, symptoms referable to the cerebrum occur in superior vena caval thrombosis. Headache is by far the most prominent manifestation and, as with other clinical manifestations, is usually aggravated by lying down and relieved when the patient is in the upright position. Immediate relief of the headaches is generally secured by venesection, which undoubtedly diminishes the pressure in the tributaries of the superior vena cava. Headache not only is aggravated by assuming the horizontal position, but is also characteristically aggravated when stooping.

Vertigo is frequently associated with headaches, but occurs less frequently. It is particularly likely to be caused by stooping.

Somnolence is an occasional symptom. Waterfield's[58] patient often fell asleep while walking or while waiting on customers. In this patient somnolence apparently came in attacks. Green's[95] patient was semistuporous for some time before death. He, however, could be aroused. The patient reported by Rijo[55] complained of insomnia especially after the ingestion of wine, during which time he had a fullness in his head. Another case with insomnia was reported by Chiray and Lebon.[86]

Prominence of the eyes may be a conspicuous manifestation and is undoubtedly due to the increased venous pressure. Staring eyes have been reported by Rees[27] and by Ehrlich, Ballon, and Graham.[1] Sudden ap-

pearance of exophthalmos was reported by Merlin.[54] Disturbed vision and papillary edema may occur, and every patient with superior vena caval obstruction should have a careful ophthalmoscopic examination.

Auditory manifestations consist of tinnitis and deafness. These symptoms, as many others, are due to venous stasis and usually disappear following establishment of adequate collateral circulation.

As a result of the venous stasis there occurs an increase in the cerebrospinal fluid pressure. Weed and Hughson[115] showed that the cerebrospinal fluid pressures are dependent largely upon venous pressures. Cerebral irritability may occur in those cases with increased intracranial tension. Psychosis has been reported by Samek.[72] Increased venous pressure frequently may result in epistaxis. In the case reported by Waterfield[58] the epistaxis was profuse, but following each attack of bleeding the patient was relieved of his symptoms. In a case reported by Samek[72] epistaxis was particularly marked upon arising in the morning.

Swelling of the face, particularly if the patient has been in the horizontal position for a long time, is a prominent manifestation. Characteristically, as in edema of the upper part of the body, swelling decreases when the patient is up and about. In our Case 1 the patient made the observation that following venous pressure determinations within three hours there was a definite decrease in the swelling of the face. Following removal of 100 c.c. of blood it was quite evident that such a decrease in the swelling of the face did occur.

During the height of the obstruction before collateral circulation has developed, these patients are likely to assume the orthopneic position as described by Jaccoud.[116] According to that author the position consists of sitting up and arching the trunk slightly forward. He states that the two conditions in which this position is likely to be found are intrathoracic tumor with superior vena caval obstruction and mediastinitis.

Dysphagia is occasionally complained of and is due either to the associated mediastinal lesion or to mediastinal edema with pressure on the esophagus. Superior vena caval obstruction per se should not produce dysphagia.

In all patients surviving long enough, collateral circulation develops. The clinical manifestations in individual cases are dependent upon and directly proportionate to the rapidity of the formation of the thrombosis and the development of the collateral vessels. In those cases in which the thrombotic lesions develop slowly and in which sufficient time has elapsed for the development of adequate collateral circulation, there are relatively few symptoms. Edema, swelling of the face, cyanosis, or other manifestations which are dependent upon and the result of increased venous pressure may be absent. Hallett,[117] in 1848, gave an excellent review of the collateral circulation developing in superior vena caval obstruction

and stated: "The channels selected are shortest and most direct." Fischer[22] in a comprehensive article divided superior vena caval obstructions into three types: (1) those in which the obstruction was only below the azygos orifice. In 166 cases, 31 were of this variety. (2) Those extending up to the azygos orifice, 121 cases; (3) those only above the azygos orifice, 14 cases. Carlson[107] in experimental work in dogs showed that ligation of the superior vena cava above the azygos was well tolerated, but that ligation of the superior vena cava below the azygos could not be done unless performed in two stages. He[107] described the collateral circulation in a minute manner, and the reader is referred to his publication for a résumé of the subject. The collateral veins vary considerably according to the patency or closure of the azygos orifice. They may be divided into the superficial and the deep. The main collateral vessels are, according to Carlson[107]:

"*Obstruction Above the Azygos Vein.—Superficial veins.* Tributaries of the axillary vein, chiefly the thoraco-epigastric vein, join a plexus of superficial veins over the thorax and abdomen. Superficial veins of the neck anastomose with those of the thorax, but these collateral veins are not prominent in the dog. The return flow of blood from the superficial veins occurs largely through the intercostal veins. Communications with the superficial epigastric and superior and inferior epigastric veins permit a return of blood through the femoral and external iliac veins, but the path through the external iliac veins is not well developed when the obstruction is above the level of the azygos vein.

"*Deep veins.* (a) The internal mammary vein, a tributary of the innominate vein, is an important vessel in the collateral circulation (Fig. 8). Anastomoses with the intercostal, anterior mediastinal and superior epigastric veins permit the return flow of blood to the heart.

"(b) The veins of the mediastinum form a prominent plexus composed of the anterior mediastinal veins, a group of posterior mediastinal veins and the pericardiac veins. Below they enter the superior phrenic veins, tributaries of the inferior vena cava.

"(c) A group of veins along the vertebral column, especially the internal vertebral plexus, communicate, above, chiefly with the dural sinuses and the vertebral veins and, below, with the intervertebral and intercostal veins.

"(d) There is a plexus of deep veins of the back formed chiefly by posterior rami of the intercostal veins. Descending branches of the transverse cervical and transverse scapular veins also appear to enter into the formation of this deep dorsal group.

"(e) The lumbar, abdominal, and suprarenal veins communicating with the superficial and deep veins of the back and with the abdominal wall empty into the inferior vena cava. The lumbar veins also join the azygos system through the ascending lumbar veins.

"(f) The superior intercostal veins, other intercostal veins, and the accessory hemi-azygos and azygos veins form an important channel of collateral circulation.

"In this type of obstruction (the superior vena cava being obstructed above the level of the azygos vein) the azygos vein with its tributaries becomes markedly distended and is the principal venous trunk through which blood returns to the heart from the upper part of the body.

"*Collateral Circulation After Obstruction of the Superior Vena Cava, Including the Azygos Vein.—Superficial veins.* The superficial veins are prominent over the chest and abdomen, beginning in the axilla chiefly with the thoraco-epigastric veins and terminating below in the superficial epigastric tributaries of the femoral veins. The superficial veins anastomose freely with the deeper veins.

"*Deep veins.* (a) The internal mammary and the superior and inferior epigastric veins form a path from the innominate vein above to the external iliac vein below.

"(b) The pericardiac veins and the anterior and posterior mediastinal veins form a well-developed plexus of collateral veins traversing the mediastinal space.

"(c) The vertebral venous plexuses are extensively developed, particularly the internal vertebral plexus, which may be traced clearly from the cervical to the lumbar region. The intervertebral, intercostal and lumbar veins anastomose with these plexuses.

"(d) The deep collateral veins of the back are present, but are not prominent. They may be traced down to the lumbar region.

"(e) The suprarenal, lumbar and abdominal veins of the dog communicate with the deep and superficial veins of the lumbar region and enter the inferior vena cava.

"(f) The azygos system, being obstructed, is diminished in size, and the flow of blood through it is apparently reversed.

"The differences between the paths of collateral circulation in the two types of obstruction may be summarized briefly. In the first type the azygos vein and its tributaries form the chief venous trunk for the return flow of blood from the upper part of the body to the heart. The lower abdominal collateral veins are relatively unimportant. In the second type of obstruction the superficial and deep abdominal vessels and the vertebral plexuses are of much greater importance. As the azygos veins are blocked, all of the blood returns to the heart through the inferior vena cava."

Wagner,[101] investigating on human cadavers, superior vena caval obstructions above the azygos and below the azygos, made the following observations: In those cases in which the obstruction was above the azygos the flow was through the renals, the suprarenals, the spermatic, and the internal iliac to the inferior vena cava. When, however, the obstruction was below the azygos, the flow was in the reverse direction,

i.e., from above downward, into the inferior vena cava. In the latter instance the superficial veins of the dorsal side of the thorax were more prominent than in the former. Based upon these investigations, he concludes that the presence or absence of dilated superficial veins of the dorsal thorax is of diagnostic value; i.e., when they are present, the obstruction is below the azygos.

In those cases in which there is a dilatation of the superficial vessels of the thorax and the abdomen there is a reversal of flow of blood, i.e., from above downward, which can be readily demonstrated. In order that reversal of flow can occur in the azygos and internal mammary vessels, it is necessary that the dilatation and venous hypertension be great enough to overcome the normal valves. According to Wagner[101] in superior vena caval obstruction the frequency of the development of collateral veins is as follows: anterior thorax, 70 per cent; neck, 64 per cent; abdomen, 47 per cent; upper extremity, 23 per cent; posterior thorax, 12 per cent. According to Chiray and Stieffel[102] varicosities along the costal margin, particularly on the left side, are of diagnostic importance in superior vena caval obstruction. Duchek[21] gives an excellent summary of the collateral circulation in superior vena caval obstruction. An insufficiently considered collateral route is through the esophageal, the gastric coronary veins, and the portal, to the inferior vena cava. At the upper end the esophageal veins communicate with the azygos. As a result of chronic, passive congestion in the kidneys because of the collateral circulation through the renal vein, albuminuria is a prominent manifestation in superior vena caval thrombosis.

The collateral veins and other manifestations of superior vena caval thrombosis are likely to be aggravated by exercise. In our Case 1, however, exercise, particularly dancing, was followed by decrease in the edema of the face and the neck, which was undoubtedly due to the milking action of the thoracic musculature. According to Milles[118] venous return is aided by skeletal muscular action. Phlebitis in the dilated collateral veins can occur, as evidenced by thrombosis of the veins in the upper right thorax in Broca's case.[94]

Alterations in voice have been reported by Oulmont,[32] and in all probability are due to the lesions which produce the superior vena caval thrombosis and not to the thrombosis itself.

Paresthesia of the upper extremity has been reported. Paresthesia of the hands and arms was present in Rijo's[55] and Riutinga's[42] cases.

*Prognosis.*—Prognosis in superior vena caval obstruction, as mentioned before, depends upon the relative rates of formation and the extent of thrombosis and collateral circulation. Cases have been reported in which there were few or no symptoms because the collateral veins developed as fast as the interference with the normal flow through the superior vena cava. On the other hand, a rapidly progressing process can produce death in a short period of time.

In the case reported by Waugh[85] the patient lived for four weeks after the onset of symptoms. Wagner[101] and Picot[81] consider the prognosis as very grave; whereas Le Tohic[50] is not so pessimistic. According to Oulmont,[32] death usually occurs in from one to six months. Balzer,[74] Beevor,[105] Comby, Vigouroux, and Collet[88] report cases of patients surviving fifteen years. Martin, René, Rouëssé, and Fiocconi[96] report a case of a patient surviving seventeen years, and Claverie[103] reports a patient surviving thirty-one years. As suggested by Carlson's investigative work, mentioned above, the prognosis depends upon the location of the obstruction, i.e., whether it is above or below the azygos.

In the 120 cases which we collected from the literature 85 patients died, a mortality of 75.9 per cent. Of those cases in which the superior

Chart 7.—Chart showing the mortality rates in superior vena caval thrombosis according to various etiologic factors.

vena caval thrombosis was the result of aneurysm, the mortality rate was 73.3 per cent; whereas in the group in which it was produced by neoplasia the mortality rate was 94.4 per cent (Chart 7). There were 24 cases in which a mediastinitis was responsible for the superior vena caval thrombosis. Of this group, 22 died (78.5 per cent) and 6 (21.4 per cent) lived. Five had an idiopathic mediastinitis with an 80 per cent mortality; 1, pyogenic mediastinitis with a 100 per cent mortality; 1, traumatic with 100 per cent mortality; 10 had a tuberculous mediastinitis with a 70 per cent mortality, and 11 had luetic mediastinitis with 81.8 per cent mortality. Forty-four had phlebitis, of which 32 (72.7 per cent) died and 12 (27.2 per cent) lived (Chart 7). There were 7 with pyogenic phlebitis, of which 57.1 per cent died; 1 with traumatic phlebitis with 100 per cent mortality. There were 4 with a tubercu-

lous phlebitis with 100 per cent mortality. There were 12 with a luetic phlebitis with a 75 per cent mortality. There were 20 with an idiopathic phlebitis with a 70 per cent mortality.

*Diagnosis.*—The diagnosis of superior vena caval obstruction is not difficult if one will only consider the condition. A patient presenting edema and cyanosis of the head, neck, and upper extremities with a normal lower half of the body should be suspected. If, in addition to this, there are superficial varicosities over the anterior thorax, particularly along the lower costal border and more improvement on the left side, the diagnosis is more likely. An important diagnostic sign is normal venous pressure in the leg and venous hypertension in the arms. Roentgenography of the mediastinum is of value for the elimination of lesions which compress the superior vena cava without producing thrombosis. Lian and Abaza[106] believe that elevated venous pressure together with a normal circulatory speed occurs in cases of superior vena caval obstruction, whereas the same increase in venous pressure associated with a decreased circulatory speed is indicative of cardiac failure, particularly when the increase in venous pressure is generalized. On the other hand, Arrillaga and Taquini[113] believe that in mediastinal compression of the superior vena cava the circulatory speed is less than normal. Reversal of flow in the superficial vessels is of diagnostic importance and can be easily demonstrated.

*Treatment.*—The treatment of superior vena caval thrombosis in the past has been very unsatisfactory as judged by the high mortality rate. In those cases in which there is a rapid progression of the thrombosis so that the collateral circulation has little time to develop, the clinical course is also rapid. Bloodletting is frequently of great value and may be life-saving. The removal of surprisingly small amounts of blood frequently relieves patients for relatively long periods of time. In our own Case 1, the removal of only enough blood incident to the determination of the venous pressure produced considerable relief for a period of approximately forty-eight hours. Waterfield[58] was able to relieve Biot's respiration in his patient by bleeding. Pastau[91] reports a case in which very large venesection apparently was life-saving. The amount of blood lost was so great that it required nine weeks for the patient to recover. He lived and was fairly comfortable for four years before his death. Rijo[55] reports a case in which the patient obtained relief by bleeding himself, which he had done for some time previously for hypertension.

Irradiation of the chest has been used because of the possibility of malignant tumors. A case is reported by Davies[44] in which it was used and was of no value. Our own Case 1 was treated by means of x-ray because of the presumed presence of a thymoma. It is important to warn against the promiscuous use of x-ray, because, although irradiation

is of distinct value in certain neoplasia, it can be harmful. Cupping of the thorax was used by Barth[20] and Broca.[94] The French have used leeches with indifferent results. Sergent and Combien[49] used unsuccessfully cutaneous cauterization. Antiluetic treatment has been given extensively, and in those cases of early lues, whether a luetic endophlebitis or mediastinitis, a great deal can be accomplished. Beneficial results following the use of antiluetic therapy have been reported by Taylor,[100] Merlin,[54] and Picot.[81]

Mediastinotomy as a decompressive measure has been advocated by Skillern[84] and Ehrlich, Ballon, and Graham.[1] These authors apparently recommend mediastinotomy as a decompressive measure in those cases with tumor. From the results obtained in our Case 1, we believe that mediastinotomy is a definite therapeutic aid in those cases in which the thrombosis is the result of a cicatrizing mediastinitis, because by releasing the cicatrix around the superior vena cava the canalization of the thrombus is facilitated and favored. According to Duval,[119] "It is a well-recognized fact that all antemortem clot which undergoes organization and subsequent canalization will in time result in the complete reestablishment of the normal circulation. In those instances in which there is organization of the thrombosis without subsequent canalization, the contraction of the replacement fibrous tissue is so great as to interfere seriously with the continuous life of the organization tissue including the newly formed blood vessels. Such contraction leads to degenerative changes for the entire replacement tissue (fibrosis). Following the degeneration there sooner or later occurs death and deposition of lime salts which produces for the entire organization tissue a lith-like hardness; i.e., calcification. In this calcified new tissue it is not possible to have canalization, because of the two factors, tissue death and strong rigidity of the calcified mass."

## DISCUSSION

The mortality rate in the reported cases of superior vena caval thrombosis has been extremely high (75.9 per cent) (Chart 7). Of importance, however, is a consideration of the morbidity rate, because in those cases in which sufficient collateral veins develop following complete closure of the superior vena cava, the patient may not die, but become a chronic invalid. Therapeusis in cases of superior vena caval obstruction has been unsatisfactory, except in those cases in which a luetic mediastinitis or endophlebitis has been energetically treated by antiluetic therapy early in the course of the disease. Mediastinotomy has been suggested in cases of tumor with the idea of decompressing the anterior mediastinum. Because of the results which we obtained in our Case 1, we are of the opinion that mediastinotomy is of value in cases of mediastinitis in which there is a cicatricial narrowing of the vena cava even though it may be thrombosed. The improvement which occurred in our patient

following mediastinotomy and extirpation of the cicatrizing connective tissue around the superior vena cava we believe is due to the opening of the newly formed vessels in the canalized thrombus. The fact that no improvement occurred in this patient during the three years prior to the mediastinotomy and that considerable improvement followed the removal of the perivenous scar is indicative that the vascular channels in the thrombus, which must have been present for some time, have been able to function only since the release of the constricting mass. The course of events in our particular case is discussed by Dr. Duval and is illustrated diagrammatically in Figs. 13, 14, and 15: "Following the vena caval thrombosis there was organization which eventually completely replaced the antemortem clot (Fig. 13, No. 2). The newly formed blood vessels which accompanied the fibrosis remained as nonfunctioning canals, because of the external pressure upon the involved portions of the vena cava by the encapsulating perivascular cicatricial tissue (Fig. 14, No. 4). This pressure undoubtedly kept them collapsed or prevented blood from passing into them as would have occurred had there not been this external pressure. To explain the subsequent partial reestablishment of the vena caval circulation, there undoubtedly was a release of this cicatricial tissue. The surgical removal of the encapsulating cicatricial mass relieved the external pressure, following which the collapsed new blood vessels in the organized or fibrotic intracaval cord became dilated by the oncoming circulation aided by the venous hypertension (Fig. 14, No. 5)." The two cases presented in the present communication represent rare heights of superior vena caval thrombosis, as in the 120 cases reported in the literature only 1 previous traumatic mediastinitis was reported, and only 1 case of pyogenic mediastinitis. We believe that our Case 1 is of the former type, whereas the Case 2 is of the latter type. It is our opinion that as a result of the injury which our first patient (Case 1) sustained there occurred hemorrhage into the mediastinum and probably a break in the wall of the superior vena cava (Fig. 13, No. 2). Organization occurred within the vessel, but because of the marked perivenous fibrous reaction, dilatation of the newly formed canals in the organizing thrombus of the superior vena cava was not possible (Fig. 14, No. 4). Only after decompression of the superior vena cava by removal of the dense, constricting, cicatricial tissue could the small veins in the organizing clot dilate (Fig. 14, No. 5).

## SUMMARY AND CONCLUSIONS

1. Superior vena caval obstruction is rare. A total of 120 cases has been collected from the world literature.

2. Forty-four (36.6 per cent) were the result of phlebitis; 35 (29.1 per cent) were caused by external compression; 28 (23.3 per cent) resulted from mediastinitis, and in 13 (10.8 per cent) the cause was unknown (Chart 1).

3. We are adding 2 new cases to the above collected group, both of which are examples of superior vena caval thrombosis associated with mediastinitis, one the result of trauma and the other the result of pyogenic infection.

4. Of all the cases of superior vena caval thrombosis, 33.3 per cent were either directly or indirectly caused by syphilis, 11.6 per cent by a tuberculous process, 8.3 per cent by heart disease, 6.6 per cent by pyogenic infection, and 1.6 per cent by trauma (Chart 5).

5. The pathognomonic signs and symptoms are:

a. Edema and cyanosis of the face, neck, and upper extremities
   (1) Aggravated by assuming the horizontal position
   (2) Relieved when erect
b. Venous hypertension in arms
c. Normal venous pressure of lower extremities
d. Development of subsequent varicosities on anterior thorax
e. Development of deep collateral vessels.

6. The mortality rate in the reported cases of superior vena caval thrombosis is high. For the entire group it was 75.9 per cent. In the group caused by neoplasia it was 94.4 per cent; in those produced by mediastinitis 78.5 per cent; those produced by aneurysm 73.3 per cent, and those the result of phlebitis 72.7 per cent (Chart 7).

7. Antiluetic treatment should be used in all syphilitic cases and particularly early. Mediastinotomy is of value not only in those cases in which there is a tumor in the anterior mediastinum, but also in those cases in which there is a cicatrizing mediastinitis with compression of the superior vena cava. In Case 1 of our reported cases definite improvement followed the removal of the constricting, cicatrizing tissue, and the mechanism by which this occurs is discussed.

CASE 1.—Miss P. S., aged twenty-three years, referred to the Tulane Hutchinson Memorial Clinic on Nov. 26, 1935, by Dr. A. F. Brock.

*Clinical Complaint.*—Varicosities of the chest, cords in the neck, and fullness of the face.

*Present Illness.*—Began three years ago when patient noticed a few varicosities on the anterior thoracic wall shortly after she injured herself while opening a bottle of coca cola. The injury occurred while she was holding the bottle over her head with the cap hooked over a transom and pulling almost hard enough to raise her weight from the floor when the cap came off and the bottom of the bottle struck her over the superior part of the sternum. She immediately became dyspneic, and ecchymoses appeared at the site of injury. After these disappeared, she noticed anterior thoracic varicosities and enlarged cordlike veins in the neck. Six weeks later she noticed that her face became full and rounded. She thought this was possibly due to a gain in weight. Before this time her cheek bones had been rather prominent, and facial fullness had not been observed even after increase in weight. The varicosities became more prominent, and her family physician made a diagnosis of disease of the thymus gland. She was given x-ray treatments in 1933, the dosage being "⅔ skin erythema with a 10 inch gap machine." The varicosities seemed to progress with each treatment, but were limited to the anterior

chest wall. Veins of the arms became more prominent; however their size was not constant. Her face became very puffy, the eyelids edematous, and from September,

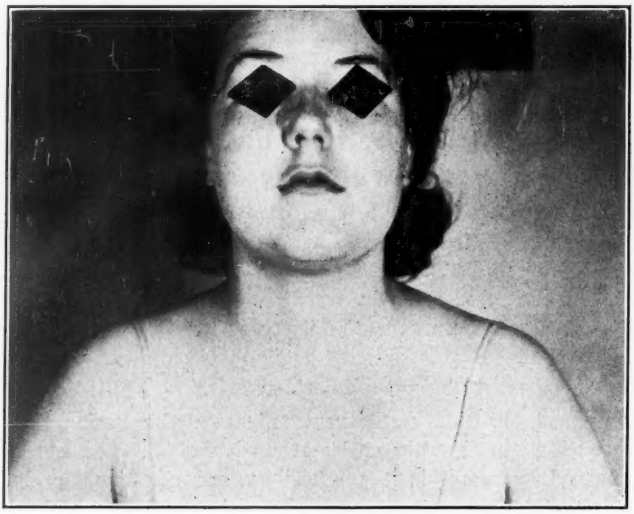

Fig. 1.—Case 1, showing swelling of face, neck, and shoulders. The edema is slightly more marked on the right side.

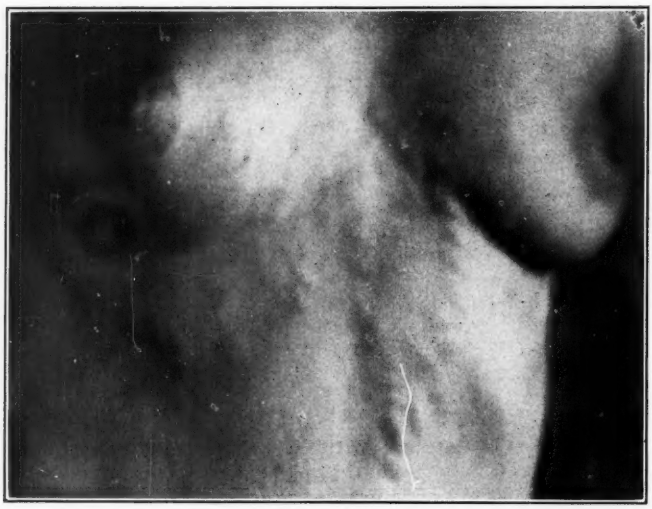

Fig. 2.—Case 1, showing varicosities of the superficial thoraco-abdominal veins, particularly marked along the left costal margin.

1933, to January, 1934, the edema of the eyelids was the most prominent symptom and was especially marked in the mornings. This condition gradually grew worse over a period of a year and a half and then remained stationary until the latter part

of 1935, since which time there has been an aggravation of symptoms. Consumption of alcohol or strenuous exercise seemed to improve the condition temporarily. Sleeping without a pillow produced a tight feeling in the neck and the head. She slept on three pillows. The swelling of the face and the neck diminished during the day, being most prominent upon arising. She thought her symptoms were slightly worse during menstruation. She felt perfectly well otherwise.

*Review of Systems.*—General health very good. Eye, ear, nose and throat: has had several attacks of epistaxis, the last one being about a week ago. Respiratory, gastrointestinal, and genitourinary tests entirely negative.

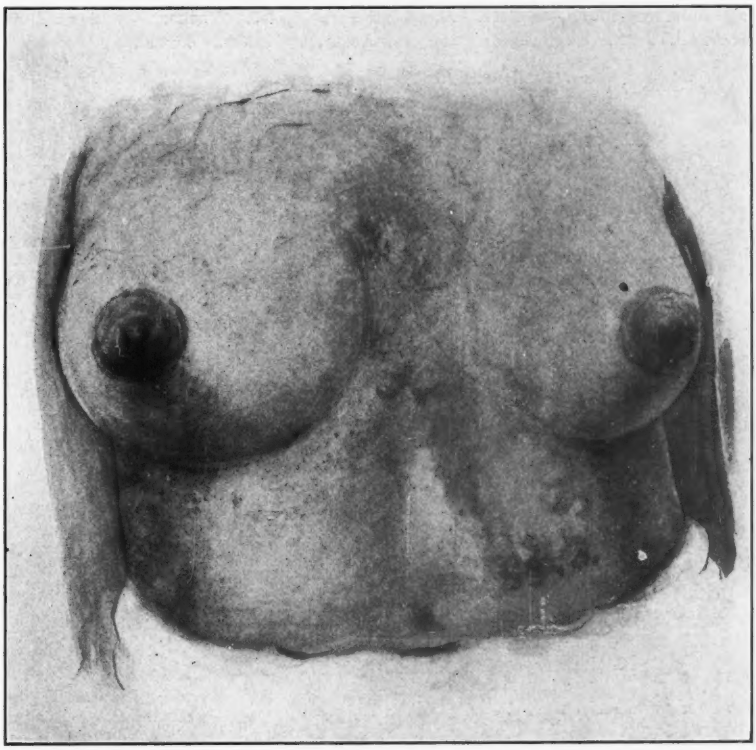

Fig. 3.—Drawing showing varicosities along both costal margins, particularly marked on the left, with development of collateral veins on the anterior portion of the thorax.

*Past History.*—Ruptured appendix with peritonitis in 1928. Two years ago the patient had a tonsillectomy performed, complicated by postoperative hemorrhage.

*Social History.*—The patient is above average in intelligence and is more or less accomplished in music. She lives with her mother in fairly good surroundings and before the depression was financially comfortable.

*Family history* is negative.

*Physical Examination.*—The patient is a white girl, twenty-three years of age, cooperative, and of average slender stature. The face is suffuse, somewhat puffy, and the right side is slightly larger than the left (Fig. 1). This is apparently due

to an edema. When in the supine position the lobes of the ears and the lips are slightly cyanotic. The neck is thick and superficial veins are engorged and under tension. The veins of the upper extremity are prominent, and there are marked varicosities over the anterior chest wall, most marked just beneath the left breast. The superficial vessels mentioned are apparently under great tension. The vessels of the conjunctiva are somewhat injected with no disturbance in the vision and no exophthalmos. The eye grounds show definitely engorged veins and old healing hemorrhage of the macula, presenting a picture of macular degeneration. There is an absolute central scotoma of the left eye for both form and color, which is more extensive to the temporal side. There is some edema extending upward from the neck and including the face. Blood pressure 118/60. The substernal notch is obliterated. The lungs show nothing abnormal. The heart is normal.

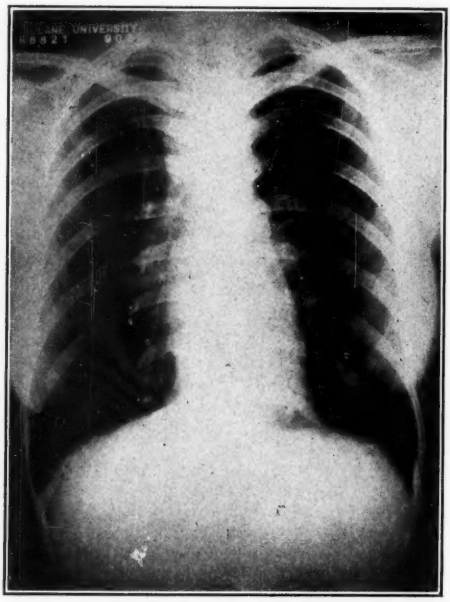

Fig. 4.—Roentgenogram, Case 1, showing no evidence of tumor formation in the anterior mediastinum. It shows, however, a shadow on the right border in the upper sternal region.

*Abdomen.*—No abnormalities aside from marked subcutaneous varicosities just beneath the left costal margin (Figs. 2 and 3).

*Pelvis.*—No abnormalities.

*Extremities.*—Both upper extremities are swollen. The right shoulder and arm are larger than the left. Superficial veins of the arms and the axilla are prominent.

*Clinical Diagnosis.*—Obstruction of superior vena cava.

*Laboratory Findings.*—Electrocardiogram negative. No fixation of electrical axis.

X-ray examination reveals a shadow in the superior mediastinum to the right side of the sternum, which extends a little farther to the right than normal. The right border of this shadow is a straight line, and it is the opinion of Dr. Menville, the roentgenologist, that this is possibly a dilated superior vena cava (Fig. 4).

Wassermann is repeatedly negative.

Dec. 17, 1935: Admitted to the Tulane Surgical Service, Touro Infirmary.

Dec. 23, 1935; Venous pressure readings: right arm, 380 mm.; left arm, 350 mm.; and right leg, 120 mm. of saline. All venous pressure determinations were made directly and under basal conditions with the patient supine and with the respective vessel at the cardiac level.

Consultation with Dr. I. I. Lemann: Superior vena caval obstruction due to fibrous adhesions in anterior mediastinum.

Dec. 24, 1935: Patient had a profuse epistaxis on the left side and lost approximately 100 c.c. of blood.

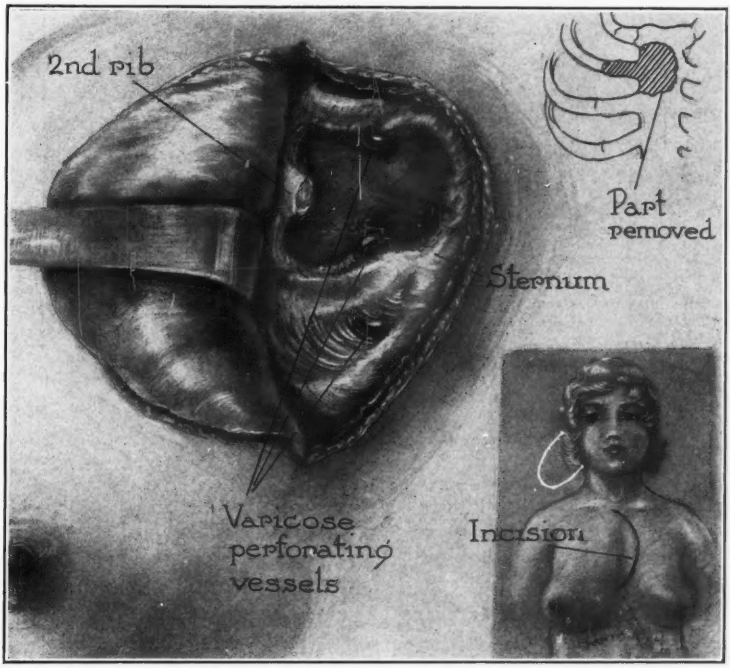


Fig. 5.—Drawing of mediastinotomy operative procedure. In the insert in the lower right-hand corner the incision is outlined, which extends from the clavicle to the left of the sternum and ends in the fourth intercostal space on the right. The upper left-hand sketch shows that the skin and the pectoralis muscles are retracted laterally, and the large, perforating varicose vessels are ligated. The insert in the upper right-hand corner shows the portion of sternum, second costal cartilage, and adjacent portion of second rib removed.

Dec. 30, 1935: *Preoperative Diagnosis:* Superior vena caval obstruction due to traumatic cicatrizing mediastinitis.

*Postoperative Diagnosis.*—Superior vena caval thrombosis; traumatic cicatrizing mediastinitis.

*Operation.*—Exploratory superior mediastinotomy and phleboclysis.

*Operative Procedure.*—Under endotracheal cyclopropane anesthesia (Waters) a curved incision is made with concavity to the right. The upper limb of the incision extends along the medial portion of the right clavicle, the extreme left of the in-

cision extends to the left edge of the sternum and the lower limb of the incision extends into the fourth intercostal space (Fig. 5). There is considerable bleeding encountered due to marked varicosities of the skin which required careful clamping, cauterization, and ligation. The flap thus produced is immobilized laterally toward the base, separating the pectoral muscles from the chest wall (Fig. 5). The second costal cartilage and anterior 2 cm. of the second rib are resected sub-

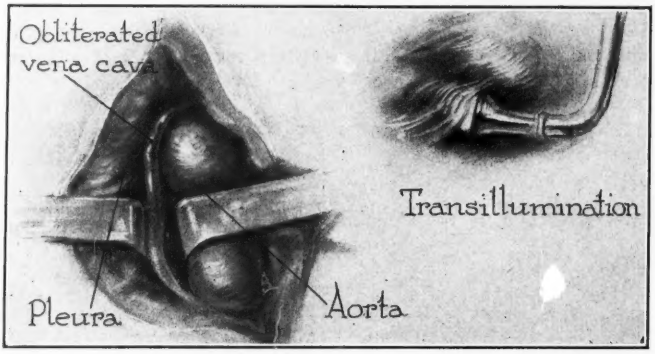


Fig. 6.—Drawing showing operative exposure of the thrombosed vena cava. At the left-hand side, after retraction of the aorta to the left and the right pleural cavity to the right, the obliterated vena cava is shown as a dense fibrous cord. The insert in the upper right-hand corner shows the method of division of the cicatricial mass, using a light for transillumination.

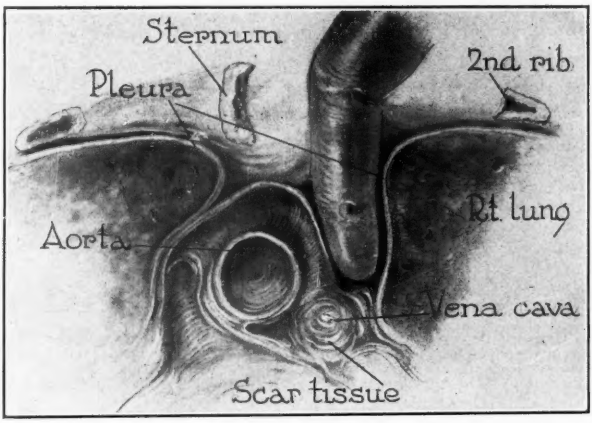


Fig. 7.—Drawing showing cross-section looking from the patient's head. By means of the finger the right pleura and the right lung are retracted to the right. The vena cava is seen to be completely obliterated and surrounded by a dense constricting scar.

chondrally and periosteally. The adjacent portion of the manubrium and the sternum are rongeured away to within 0.5 cm. of the left border of the sternum (Fig. 5). The right pleura is retracted laterally, gaining access to the anterior mediastinum. The aorta is exposed with some difficulty because of dense adhesions in the anterior mediastinum extending from the base of the heart upward. These adhesions are carefully divided by sharp dissection, using a light for transillumination before

their division (Fig. 6), with considerable difficulty, because of the dense surrounding cicatrix. The superior vena cava which occupies a more posterior position than normal is exposed. The posterior displacement of the cava is apparently due to the contraction of the encasing dense scar (Fig. 7). The vessel is exposed from the junction of the two innominate veins down to the entrance into the right auricle. Instead of the normal-sized, thin-walled vessel, it is completely obliterated and is transformed into a dense, fibrous cord approximately 5 mm.

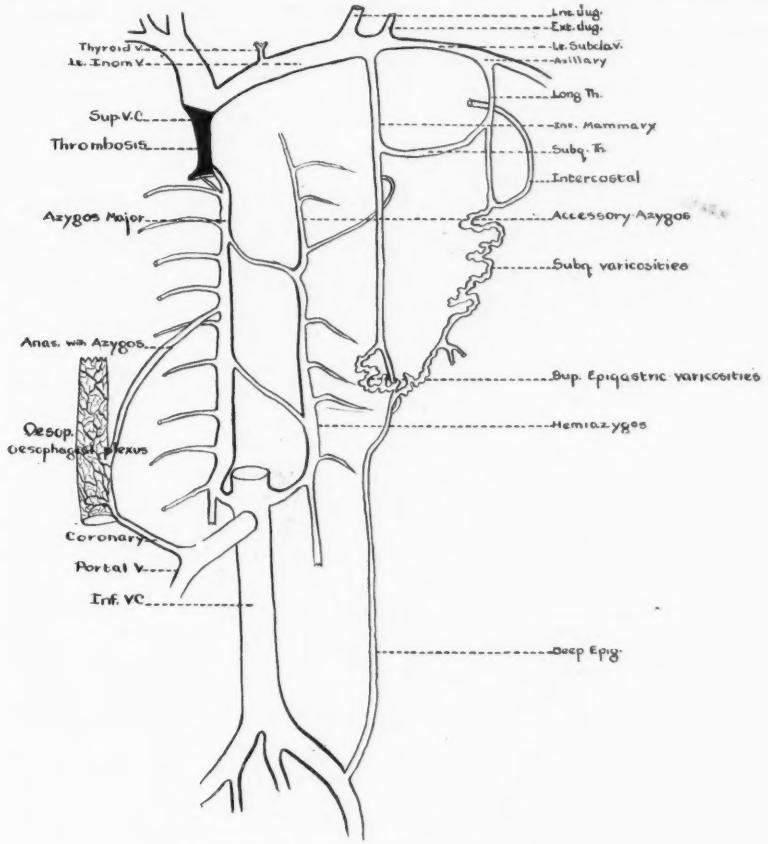


Fig. 8.—Diagrammatic drawing showing obstruction of the superior vena cava in Case 1, in which the entire vena cava from the junction of the two innominate veins down to its entrance into the auricle is thrombosed. The collateral veins developed in this case and the potential collateral vessels are shown. It is seen that the superficial varicosities extend between the long thoracic vein on the left to the superficial epigastric. The blood from the upper extremities, the head, and the neck is shunted through these collateral veins into the tributaries of the inferior vena cava.

in diameter (Figs. 7 and 8). A needle is introduced into the obliterated vein and aspiration attempted, but nothing obtained. The dense cicatricial tissue overlying and on either side of the obliterated superior vena cava is removed, thus relieving any external constricting influence in the vessel. Before closing the wound,

it is evident that the retractor holding the right pleura laterally has perforated the pleura. This is closed by means of interrupted silk sutures, the lung being inflated before complete closure. The pectoral muscles are resutured to the chest wall by means of interrupted silk, following which the fascia is closed by means of continuous suture. Skin is approximated with continuous silk. A compression sponge bandage is applied.

The patient stood the operation well, receiving 455 c.c. of whole blood postoperatively, and left the table in good condition. During the course of the operation the venous pressure in the left arm was 640 mm. of saline.

Jan. 2, 1936: Patient seems clinically improved. The venous pressure is 350 mm. in the right arm, and she has no complaint.

Jan. 6, 1936: Patient has had an uneventful convalescence. The edema is apparently less.

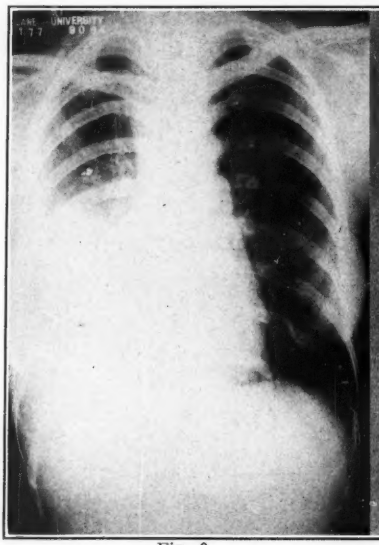

Fig. 9.

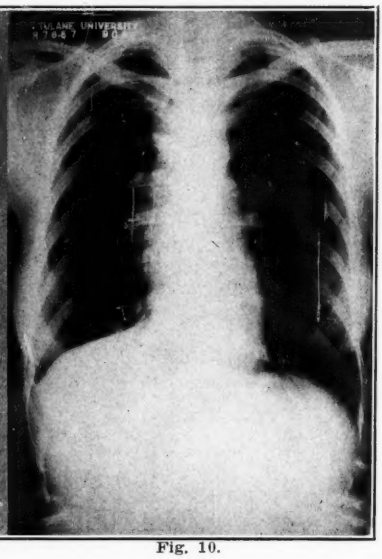

Fig. 10.

Fig. 9.—Roentgenogram, Case 1, taken two and a half months after mediastinotomy, showing elevation of the left leaf of the diaphragm and homogenous mass at the left base, which is suggestive of fluid.

Fig. 10.—Roentgenogram taken four months after mediastinotomy, showing descent of diaphragm to its normal position and no evidence of fluid in the right hemithorax.

Jan. 11, 1936: Patient discharged from the hospital.

Jan. 18, 1936: The puffiness of the face is much improved. Patient states that her vision is better in that she has not had to use her glasses for reading, whereas previously they were necessary at all times.

Feb. 17, 1936: Patient has no complaints and feels fine. The wound has healed completely. There is a definite decrease in the edema of the face and the arms. Blood pressure in the right arm is 102/68; left arm 100/70. Pulse 110. *Roentgenogram of the Chest:* The prominence of the superior vena cava as previously reported is still present. There is elevation of the right hemidiaphragm with suggestion of fluid at the base (Fig. 9). The left hemithorax is negative.

Feb. 28, 1936: An attempt is made to visualize the collaterals by the intravenous injection of 6 c.c. of thorotrast in each arm. Roentgenograms made following this were unsatisfactory.

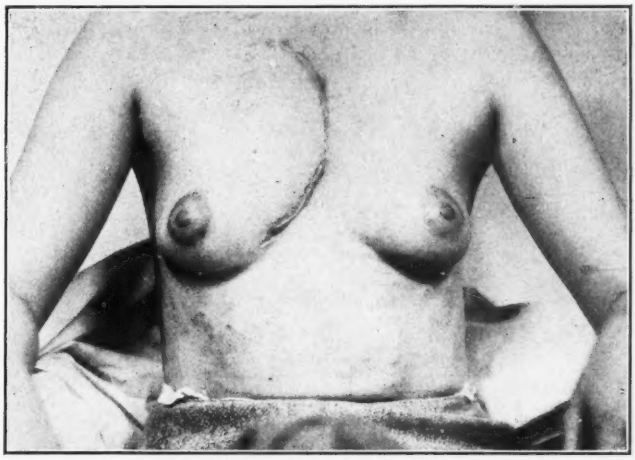

Fig. 11.—Case 1, showing operative scar and diminution in the varicosities in the lower part of the thorax and the upper part of the abdomen.

Fig. 12.—Case 1, showing decrease in the size of the face, which is especially marked when contrasted with the preoperative photograph, Fig. 1.

March 5, 1936: Patient states she has not felt so well since February 28 following a few highballs. Face and arms seem slightly more swollen. Venous pressure determinations: right arm, 320 mm.; left arm, 275 mm. of saline. In the supine position the lips assume a livid, violaceous color, which disappears when the patient stands or sits.

March 19, 1936: Patient feels fine and would like to do some strenuous exercise. The general appearance is excellent. The face seems much less puffy. However, when placed in the recumbent position, the same livid hue is present. Venous pressure: left arm, 310 mm.; right arm, 362 mm.; right foot, 60 mm. of saline.

April 21, 1936: Blood pressure 110/70 in each arm. Venous pressure: right arm, 320 mm. Patient feels fine; face becomes full and suffuse in prone position and ears become slightly livid. She states that she always feels better after venous readings in that it seems to relieve "a certain tension in the head," and that furthermore for forty-eight to seventy-two hours after venous pressure readings her face becomes much less swollen. She further claims that excess exercise in the upright position has always reduced the puffiness of the superior half of the body,

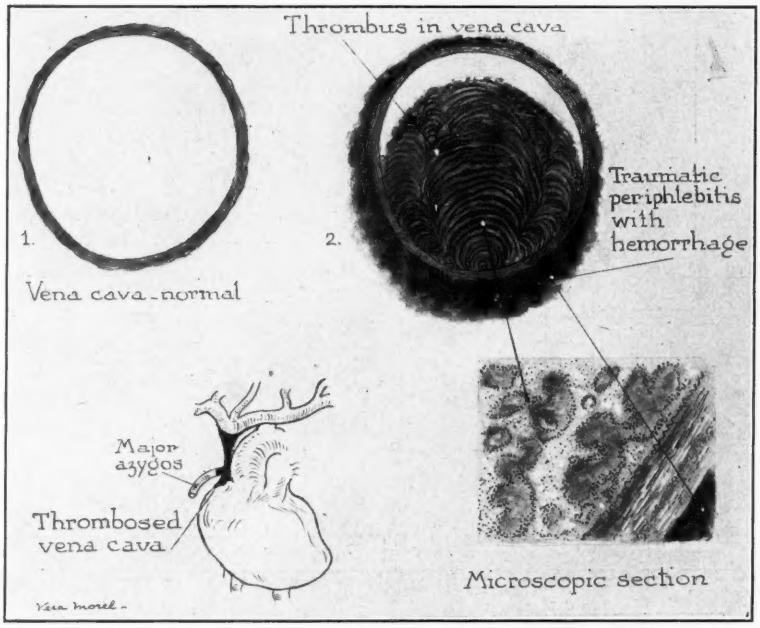


Fig. 13.—Diagrammatic drawing after Duval, showing sequence of events in the production of superior vena caval thrombosis in Case 1. Subdivision 1: Normal vena cava. Subdivision 2: Thrombus forming in vena cava as a result of trauma with probable break in endothelium at inferior vena cava. Surrounding the vena cava is hemorrhage and evidence of traumatic periphlebitis. In the lower right quadrant is a diagrammatic microscopic section of the same. In the lower left quadrant is a diagrammatic drawing of thrombosis in Case 1 showing the extent of the involvement from the junction of the two innominate veins down to the entrance of the cava into the auricle with obstruction of the opening of the azygos major.

especially the face. She has observed that there is a fluctuation in the puffiness of her face from time to time in that some days it is much better than others.

April 24, 1936: Roentgenogram: No change in mediastinal shadow previously reported. Right hemidiaphragm in normal position. No evidence of fluid (Fig. 10).

April 30, 1936: She has no complaints. The edema is becoming less with each visit, and she states that she danced from nine o'clock to eleven and from twelve to three on the previous night. The venous pressure determinations: right arm, 280 mm.; left, 276 mm.; right leg, 80 mm. (Figs. 11 and 12).

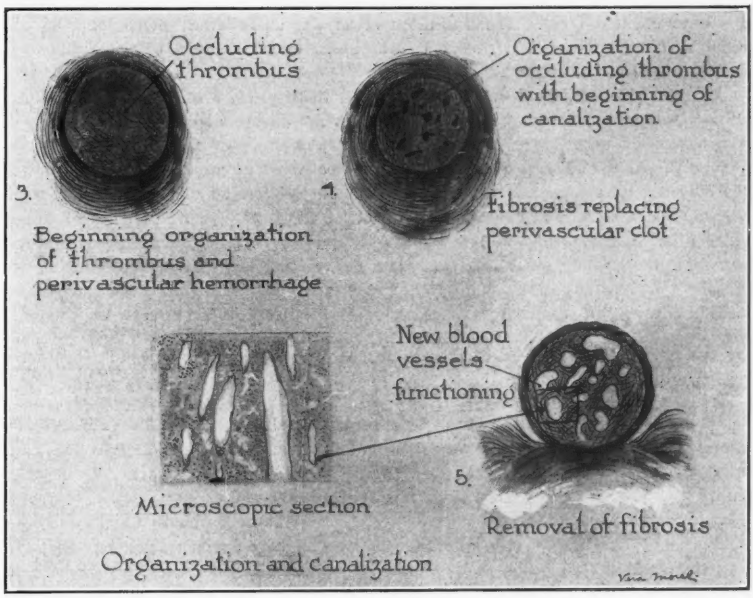


Fig. 14.—(3) Diagrammatic drawing showing complete occlusion of the superior vena cava by the thrombus. (4) Beginning organization of the occluding thrombus and very early canalization. The perivascular clot is being replaced by fibrosis. Although there has been attempted canalization, because of the constriction around the vena cava produced by the cicatrizing fibrous tissue, these vessels are unable to function. (5) Diagrammatic drawing showing the effect of removal of the upper four-fifths of the perivenous cicatricial tissue, allowing dilatation of the canals in the organized clot and permitting blood to flow through them.

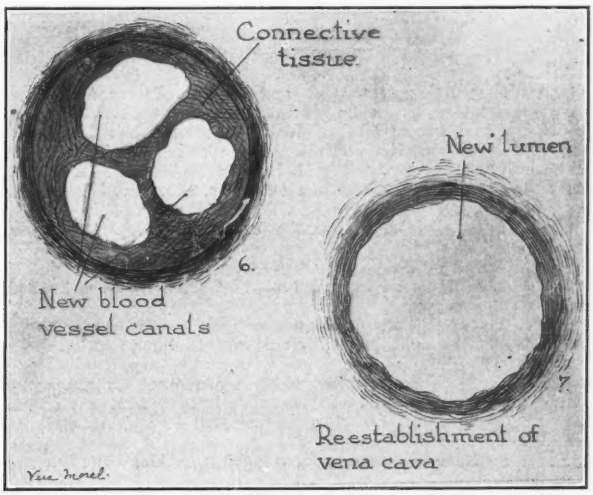


Fig. 15.—(6) Further dilatation of canals in the organized thrombus with possibility of transporting larger quantities of blood. (7) The possible complete restoration of the lumen of the superior vena cava. It is believed that the events shown in these diagrammatic drawings probably account for the improvement observed in Case 1, following the removal of the constricting perivenous tissue.

All the laboratory findings have been essentially negative, including urine, blood, Wassermann, and feces. The last blood count was as follows: hemoglobin 85 per cent; R.B.C. 4,560,000; W.B.C. 6,000; differential—S.M. 16, L.M. 5, neutrophiles 78, eosinophiles 1, color index 0.78.

Résumé: A girl, aged twenty-three years, three years previously had sustained severe injury to anterior thoracic wall. After subsidence of ecchymoses, developed varicosities on anterior surface of thorax with swelling of face, neck, and arms. Was orthopneic and had epistaxis. Symptoms were aggravated by lying down. No evidence of mediastinitis, syphilis, or tuberculosis. Had marked varicosities of anterior chest wall, particularly the left costal margin. Venous pressure of upper extremities, 350 mm., and of lower extremities, 160 mm., of saline. Mediastinal exploration because of suspected traumatic cicatrizing mediastinitis. Operation revealed superior vena cava obliterated and transformed into dense fibrous cord, 5 mm. in diameter, surrounded and constricted by firm cicatrix in anterior mediastinum. Removal of constricting fibrous scar. Progressive postoperative improvement, consisting of diminution in swelling of upper half of body, relief of symptoms, and decrease in venous pressure.

CASE 2.—This case was superior vena caval thrombosis was obtained through the courtesy of Dr. K. Brau by personal communication. Dr. Brau observed this case in Porto Rico. In 1933 a man, aged twenty years, was shot with a .22 rifle, the bullet just piercing the sternum. It was fairly easily removed by a probe the same day. On the morning of the second day the patient appeared to be convalescing without complications, in the afternoon he was slightly dyspneic, and on the third day there was evidence of cyanosis of the face, especially marked around the nose and the lips. The eyes were prominent. There was some edema of the neck, the supraclavicular region and upper chest; there was marked turgidity of the vessels of the neck. Patient had some orthopnea. The evening of the third day there were minute veins over the superior chest, which were seen without difficulty. On the fourth day all symptoms grew progessively worse, and the patient died in the evening. Necropsy showed a purulent mediastinitis, periphlebitis, and thickened walls of the mediastinal vessels including the superior vena cava, which was completely obstructed by a thrombus.

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## DISCUSSION OF SYMPOSIUM ON THE HEART, GREAT VESSELS, AND ESOPHAGUS

"WOUNDS OF THE HEART. REPORT OF TWELVE CASES," BY ELKIN.

"FURTHER DATA ON THE ESTABLISHMENT OF A NEW BLOOD SUPPLY TO THE HEART BY OPERATION," BY BECK.

"REDUCTION OF CARDIAC IRRITABILITY BY THE EPICARDIAL AND SYSTEMIC ADMINISTRATION OF DRUGS AS A PROTECTION IN CARDIAC SURGERY," BY MAUTZ.

"STUDY OF CARDIOPERICARDIAL ADHESIONS WITH REFERENCE TO OPERATION UPON THE HEART," BY HOSLER AND WILLIAMS.

"SUPERIOR VENA CAVAL THROMBOSIS," BY OCHSNER AND DIXON.

DR. HOWARD LILIENTHAL.—I should like to comment on merely two points. The first one is in regard to the beautiful operations which Dr. Elkin has reported, and since he thinks that the median section of the sternum gives the best exposure, and since he also seems to have found difficulty in making this median section, I should like to call attention to an instrument which he may not have had in his possession, and that is the sternum shears of Schumacker. This is a powerful instrument, and works so easily that it requires no effort to bisect the sternum longitudinally, or transversely if necessary. I recommend it to those of you who have the privilege of doing this work.

One other point, and that in regard to Dr. Ochsner's paper. He showed pictures which illustrated the dilated veins in the subcutaneous part of the chest wall. I have been using infra-red photography for this purpose, and I assure you that it is a valuable adjunct in following the progress of a case to see whether the veins are growing larger or smaller or more or less numerous. It is very simple, inexpensive, and the results are really most striking.

DR. ELLIOTT C. CUTLER.—It would appear that Dr. Beck's attack upon this problem of the heart inadequately supplied with blood has brought forward a method in keeping with our understanding of both the physiology and the pathology involved. It is important that when discussing this problem before this Association we should keep quite clear in our minds and make clear in our publications the reasons for such an undertaking as Dr. Beck's, since otherwise we may confuse the general practitioner who learns from our publications that some of our group are taking away the pericardium, and others, as in this instance, are putting it back on again. I fear many practitioners may think this a matter of only academic interest unless we can make clear to them all the underlying reasons for the undertaking.

I should like to ask Dr. Ochsner, who presented statistics concerning small hearts and large hearts with pericardial adhesions, whether he has any evidence that in the small heart there is additional vascularization between pericardium and myocardium, and whether the opposite condition exists with a large heart. Has he found data which would strengthen Dr. Beck's contentions that with a large heart without vascularization there is more likely to be a history of angina pectoris?

DR. LEO ELOESSER.—May I ask Dr. Beck to tell us how much time intervened between ligation of the coronaries and death of the animals?

DR. A. L. LOCKWOOD.—I should like to congratulate Dr. Beck and his associates on their courage in attempting to deal with this very serious problem, a condition that is on the increase with the life we are living at the present time.

Would he tell us about the type of anesthetic employed, how soon after operation death occurred in those who died, and apparently what was the cause, and in those who survived did he observe any sign of irritation particularly attributable to the graft?

DR. HARRY C. BALLON.—I should like to ask Dr. Beck about the choice of cases, the indications for operation; further what signs and symptoms are to be regarded as evidences of a coronary circulation beyond repair yet worthy of being helped by an operation. I was of the opinion that not a few patients were able to develop a collateral circulation for themselves. I am therefore wondering which ones, with the passing of time, are going to be able to carry on without help and which ones are going to require the benefits of an operation.

DR. EDWARD D. CHURCHILL.—The paper by Dr. Hosler was of peculiar interest, and raised a very important point, and I think we must seriously consider the evidence he has produced. If he is correct, it throws [illegible] the original concept of the Brauer operation. I am inclined to believe he is right in the majority of instances. The classic description of the heart tugging against the chest wall that hypertrophies, in order to move the bony framework with every contraction, is a vivid concept, and it is going to be one that is difficult to eliminate from the literature. Personally, I have never seen that type of case. I have done the Brauer operation for hypertrophied heart with valvular disease, but that is a different principle entirely. I should say that perhaps Dr. Hosler's statement (I have forgotten exactly how he worded it), that the effects of the Brauer operation have been exaggerated, might be modified to say that the effects of the Brauer operation have been misinterpreted, or been put on an incorrect physiologic basis.

I still believe that in an occasional case extrapericardial adhesions may cause hypertrophy of the heart. We have some autopsy evidence that points to such a conclusion.

DR. EVARTS A. GRAHAM.—Apropos of the point Dr. Churchill has just raised, I should like to say that last fall a patient was in the Barnes Hospital at St. Louis, totally incapacitated for two years, a young farmer. Roentgenkymography, which is a new and a very ingenious method of studying the heart by actually taking moving pictures of the pulsation, indicated that this man's heart was not pulsating properly on the left side. I operated on the man and found adhesions between the pericardium and the sternum, definite scar tissue. These were released, the pericardium was opened, and it was found not to be thickened; there were no adhesions between the visceral and the parietal pericardium. Two costal cartilages were removed in the performance of the operation, and that is all that was done, and the wound was closed. The man made an uneventful recovery, and he was examined later and found to have perfectly normal pulsation on the left side of his heart. This spring he has been doing almost normal work on the farm. For the first time in three years that has been possible for him.

I think there is no question whatever about the fact that there are occasional cases in which the Brauer operation is entirely justifiable as a procedure and may be expected to give good results. As a matter of fact this is the third case in my own experience in which the simple Brauer operation has had a result comparable with the one which I have just mentioned, this most recent case.

On the contrary, there are probably a great majority of cases in which the simple Brauer operation is of no particular value. It would be unfortunate, however, to condemn the Brauer procedure of cardiolysis too strongly, in the light of very definite evidence that occasionally it is of very great value.

DR. POL N. CORYLLOS.—I recall a similar case to the one reported by Dr. Graham, two years ago in New York, a working man fifty-six years of age. He had had pneumonia two years previously. In the x-ray picture, we saw that there were adhesions between the pericardium and the posterior surface of the sternum. An operation was performed, with the resection of four ribs, the second, third, fourth and fifth, with about 8 or 10 cm. removed. Nothing else was done. He made an uneventful recovery, and he is in perfect condition.

DR. CLARENCE E. BIRD.—Those of us who are connected with the large southern hospitals, charitable institutions, see most of the heart wounds collected in series. Some of the queerest things happen in these heart injuries. A bullet entering the heart on one occasion was found in the right common iliac artery or vein. It was impossible to determine which. In another case an anterior mediastinal hemorrhage occurred, which caused compression upon the heart, which simulated a cardiac intrapericardiac hemorrhage. In another patient who was seen with evidences of cerebral anoxemia, the symptoms simulated those due to injury to the skull or brain itself. Another patient following operation had a right brachial monoplegia and a motor aphasia.

There are many points of diagnosis which are extremely important, and as Dr. Arnold Griswold, in our clinic in Louisville, has brought out in a recent article on pericardiectomy, it is desirable for the house staff to be heart-conscious. That is, they must be familiar with Beck's triad, and must be able to apply it. They must also realize that wounds of the chest do not necessarily need to be directly over the precordium in order to produce a cardiac injury, and, finally, the cerebral symptoms should not always be ascribed to cranial injury.

The venous pressure is extremely important in the thorax. The other methods of diagnosis Dr. Elkin has spoken of. But another important point is continually to observe these patients; sometimes they show tamponade, and a half hour later they will not show it, and they will show it again and then will not show it. That is undoubtedly due to hemorrhage which is leaking into the pleural cavity intermittently. So the indications for operation are continuous or intermittent tamponades, or evidence of continuous hemorrhage into the pleural cavity.

Fluoroscopic examination is important, and I should think that the roentgenkymogram would be important as a means of diagnosis in these patients, except that the patients are pretty ill and the roentgenkymogram is usually taken in an upright position.

Finally, at operation, no drainage should ever be used except drainage from the pericardium into the pleural cavity. As you know, external drainage leads to various complications, secondary infection of wounds.

DR. CLAUDE S. BECK.—It does seem paradoxical perhaps, as Dr. Cutler suggested, that in one type of operation adhesions to the heart are severed and in my operation adhesions to the heart are produced. The subject of cardiac adhesions has been poorly understood, and I believe our concepts of the subject need revision. A new nomenclature is needed for the pericardial disorders. The terms that have been used are frequently meaningless and sometimes misleading. For example, what does the term adhesive pericarditis mean? It refers to an inflammatory process of the pericardium producing adhesions. It does not indicate whether the adhesions are intrapericardial or extrapericardial or both. It implies the existence of inflammation, but as commonly used the inflammatory process may have been burned out for a long time and only residual scar is present. In this respect the term pericarditis is a misnomer. It does not indicate in what way the adhesive process interferes with the circulation; indeed, it does not indicate whether the circulation is or is not in any way disturbed by the process.

I think we can separate out a number of distinct clinical entities in those cases in which pericardial infection or residual scar is present. First we have the compressed heart. The compressed heart is the exact opposite of the dilated heart. The compression may be acute or chronic. Acute compression of the heart is produced by fluid that has formed rapidly in the pericardial cavity or in the mediastinal space. Adhesions play no part in this clinical picture. Chronic compression of the heart is produced by residual scar around the heart. The infectious process may or may not have burned itself out. Adhesions between heart and scar play no part whatever in the production of the chronic compression syndrome. When present they are silent and incidental. Nevertheless, those who have written on this subject persist in using such terms as adhesive pericarditis, constrictive pericarditis, concretio cordis, Pick's disease, etc. A sterile transudate or a tumor can give you the identical clinical picture. The compressed heart is always a small shrunken organ. It cannot dilate. It cannot hypertrophy. The amount of work that a compressed heart does may be only 50 per cent of the normal. It cannot do any more work than pump the small amount of blood that happens to go through it. It is a silent heart; it produces little or no precordial activity, and the heart sounds are muffled.

Infection together with its residual scar can interfere with the heart in other ways. Residual scar can affect the circulation by angulation or by torsion of the heart. You can produce this very nicely in the acute experiment and occasionally, rarely, you find it in a patient. I have one such patient now under observation. His heart is angulated sharply toward the left axilla. In certain positions, such as stooping forward, he is all right. When he stands erect with his shoulders back, the pulse rate increases, and he gets pale and faints. I believe this patient's cardiac abnormality is due to kinking of the great vessels at the base or perhaps to torsion of the heart. For the relief of this condition thoracoplasty is indicated. Thoracoplasty will allow the heart to assume a more normal position. This must not be confused with the Brauer operation because it involves an entirely different principle from the Brauer operation.

Another possible (although questionable) way in which infection or its residual scar may affect the heart is by adhesions to the chest wall, diaphragm, etc. I do not believe that Dr. Hosler cares to say that the heart cannot be disturbed under any circumstances by such adhesions. It is almost unbelievable that this waste of energy of heart pulling on surrounding structures should not produce hypertrophy. The experiments by Hosler and Williams have not given hypertrophy. My associates and I have placed grafts of skeletal muscle, fat, pericardium, etc., on the heart in several hundred experiments and we never found cardiac failure or cardiac hypertrophy resulting from these adhesions. If this pull upon the chest wall by way of adhesions does not disturb the mechanics of the heartbeat, the principle of the Brauer operation is wrong. We have not been able to support Brauer's hypothesis by experimental work. This type of thoracoplasty has given beneficial results in only a small percentage of cases. In most of them the result was not beneficial. The good results may have been obtained not because of Brauer's hypothesis but perhaps because of the decompression given to the heart. Decompression of the large heart may be an important factor in these cases.

Dr. Graham, did you refer to cases with large heart?

DR. GRAHAM.—Not particularly large, but there was not very much decompression in the last case I mentioned. The point about decompression, however, is the factor in the Brauer operation, and is the one that I called attention to in this society about ten years ago. It would agree quite fully with what you say about it.

DR. BECK.—In general then it would appear that adhesions to the heart are silent and incidental, that the only proved way in which they can disturb the heart is by angulation and torsion of the heart.

Dr. Lockwood wanted to know about the anesthesia. We used light nitrous oxide ether anesthesia. We did not find that the grafts produced any irritation of the heart. The pericardium was left widely open.

The cases of coronary disease that I operated upon were all advanced cases. In general one should avoid patients who are orthopneic and in failure. You would prefer not to take patients who had had a recent coronary thrombosis.

A brief abstract of the cases in which operation was done is in the paper.

I should like to comment on Dr. Mautz's work. The direct application of drugs to the heart will find a place in cardiac surgery. So far as I know, this is an entirely new development in physiology and surgery. A few drops of 5 per cent procaine applied to the right auricle will stop auricular fibrillation. I have seen the heart rate increase to 180 per minute after working on the epicardium. A few drops of procaine slows the rate to about 100 per minute. Extrasystoles can be avoided in this way. I had one patient from whose heart I was resecting a compression scar, and while the dissection was in progress he developed many extrasystoles. A little later the ventricles fibrillated, the heart stopped, and the patient died. I believe we could have prevented ventricular fibrillation by the use of procaine on the surface of the heart. We had to learn for ourselves the beneficial effects of quinidine. So much confusion exists in the minds of cardiologists about this drug that we did not know whether we should or should not give quinidine. On the basis of experiments we found that quinidine is a very valuable drug in heart surgery. It steadies the heart, keeps it regular and slow, and can prevent fibrillation of the ventricles. I am using quinidine on patients both before and after operation.

DR. ROBERT M. HOSLER.—According to Moritz, Hudson and Orgain, Thorel in 1903 described a heart in which the coronary arteries were occluded and the patient died of some other cause. Adhesions were present, and Thorel suggested that the blood supply to the heart came through the adhesions. This with similar observations by Moritz led to Dr. Beck's original experiment. Dr. Beck was the first to make the observation that adhesions could and actually did transport blood to the human heart. He transected a band of adhesions extending from a compression scar about the heart to the base of the left ventricle. Both ends of this transected adhesion bled profusely.

I also wish to say that there are recorded cases of pulmonary tuberculosis in which the heart has been displaced by adhesions into the right chest or markedly to the left. Usually circulatory symptoms are absent unless angulation of the heart with distortion of the great vessels has taken place. Thoracoplasty may prove beneficial.

# The American Association for Thoracic Surgery

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## The American Association for Thoracic Surgery

The nineteenth annual meeting of the American Association for Thoracic Surgery was held at the Mayo Clinic, Rochester, Minn., on May 4, 5, and 6. The attendance was unusually good; 69 members were registered and 81 guests.

In addition to the formal papers presented there were interesting demonstrations of clinical and experimental work and operative clinics. The inability at the last moment of Dr. Sauerbruch to attend the meeting was a great disappointment.

The Officers and Council were elected as shown above.

In recognition of his contributions to the field of thoracic surgery Honorary Membership was conferred upon Dr. Franz Torek, of New York.

The resignations of the following members were accepted:

Dr. James M. Hitzrot—New York, N. Y.
Dr. J. H. Wilms—Cincinnati, Ohio

The following Active Members were retired to Senior Membership:

Dr. Hugh Auchincloss—New York, N. Y.
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Dr. Hermann Fischer—New York, N. Y.
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Dr. Urban Maes—New Orleans, La.
Dr. George P. Muller—Philadelphia, Pa.
Dr. Samuel Robinson—Santa Barbara, Calif.
Dr. George A. Stewart—Baltimore, Md.

The following Associate Members were advanced to Active Membership:

Dr. Clyde Allen—Detroit, Mich.
Dr. Dewey Bisgard—Omaha, Neb.
Dr. Herbert Carlson—Minneapolis, Minn.
Dr. Lincoln Fisher—Mansfield, Ohio.

Dr. Samuel O. Freedlander—Cleveland, Ohio.
Dr. John N. Hayes—Saranac Lake, N. Y.
Dr. Jerome Head—Chicago, Ill.
Dr. George Ornstein—New York, N. Y.
Dr. Warriner Woodruff—Saranac Lake, N. Y.

The following Candidates were elected to Associate Membership:

Dr. Henry L. Cabitt—Boston, Mass.
Dr. George M. Curtis—Columbus, Ohio.
Dr. Frederick C. Fishback—Washington, D. C.
Dr. George G. Finney—Baltimore, Md.
Dr. John H. Gibbon, Jr.—Philadelphia, Pa.
Dr. E. C. Janes—Hamilton, Ont.
Dr. N. Logan Leven—St. Paul, Minn.
Dr. Jacob James Longacre—Cincinnati, Ohio.
Dr. Edgar Mayer—New York, N. Y.
Dr. Ross McPhail—Lakeview, Wash.
Dr. Herbert Willy Meyer—New York, N. Y.
Dr. Lawrence W. Nehil—Ann Arbor, Mich.
Dr. Robert E. Plunkett—Albany, N. Y.
Dr. B. P. Potter—Secaucus, N. J.
Dr. Albert O. Singleton—Galveston, Texas.
Dr. George F. Skinner—Saint John, N. B.
Dr. Paul Ringer—Asheville, N. C.
Dr. J. A. Vidal—Montreal, Canada.

The following committees were appointed:

*Membership Committee*

Ethan Flagg Butler, Chairman

Frank S. Dolley — Norman Bethune
I. A. Bigger — Claude S. Beck

*Program Committee*

Edward D. Churchill, Chairman — Edward S. Welles
President, Secretary, Editor, ex officio.

The next annual meeting of the Association will be held in Saranac Lake, New York. The date will be announced later.

We were glad to see many guests and visitors present at the Rochester meeting. The Association welcomes any one interested at its meetings. Nonmembers having contributions of merit are invited to present these to the Program Committee (Dr. Edw. Churchill, Chairman) for consideration and also to apply to him for space in the program for discussion. Nonmembers may receive advance programs by applying to the Secretary, Dr. R. H. Meade, Jr., 2116 Pine Street, Philadelphia, Pa.

Intending contributors are informed that papers containing rows of figures are rarely of interest when read aloud. If statistical conclusions need be supported by figures, these should be presented on charts, not lantern slides, so that they may be constantly in view of the audience while listening to the speakers expounding conclusions drawn from them. Verbal repetition of these charts should be unnecessary; the charts themselves should suffice.

Intending contributors are requested to submit names, titles and detailed abstracts to the program committee at least two months before the date of the next meeting. If their papers are accepted, it is recommended, in order to have an apposite discussion, that the authors designate one or two discussers, that they get their consent to discuss, and that they send their copies of their papers early.

# CONSTITUTION OF THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

(ADOPTED MAY 10, 1927)

## Article I—Name

SECTION 1.—This Association shall be known as the American Association for Thoracic Surgery.

## Article II—Object

SEC. 1.—The object of the Association shall be to encourage and stimulate investigation and study that will increase the knowledge of intrathoracic physiology, pathology and therapy, to correlate such knowledge and disseminate it.

SEC. 2.—To attain this object, the Association shall hold at least one scientific meeting every year in which free discussion shall be featured; shall conduct a Journal for the publication of the papers presented at this meeting and other acceptable articles; shall sponsor and support a Registry for Chest Tumors; shall cooperate with other organizations working toward the same end; and shall undertake such other activities as the Council or the Association as a whole may decide.

## Article III—Membership in Congress

SEC. 1.—*As a component member of the Congress of American Physicians and Surgeons, this Association subscribes to the Constitution and By-Laws of the Congress.* (Amendment to delete this Section passed first reading 1936.)

## Article IV—Membership

SEC. 1.—There shall be four classes of members: Active, Associate, Senior, and Honorary. Admission to membership in the Association shall be by election. Membership shall be limited, the limits on the respective classes to be determined by the By-Laws. The power to vote and the privilege of holding elective office are limited to active and senior membership.

SEC. 2.—Election to Active, Senior and Honorary Membership shall be for life, subject to the provisions of Sec. 3, following. Election to Associate Membership shall be for a limited period of time, as determined by the By-Laws. During this limited period, an Associate member, if properly qualified, may be elected to Active membership. At the expiration of this limited period, an Associate member, if not yet qualified for Active membership, must either be reelected to an additional period of Associate membership, or be dropped from the rolls of the Association.

SEC. 3.—Membership may be voluntarily terminated at any time by members in good standing. The Council, acting as a Board of Censors, may recommend the expulsion of a member on the grounds of moral or professional delinquency, and submit his name, together with the grounds of complaint, to the Association as a whole at any of the regularly convened meetings, after giving the member so accused ample opportunity to appear in his own behalf.

## Article V—Officers and Government

SEC. 1.—The officers of the Association shall be a President, a Vice President, a Secretary, a Treasurer, an Editor, and five Councilors. These ten officers and councilors shall be the governing body of the Association, and shall have full power to act in all matters, except as follows:

1. They may not alter the initiation fees or annual dues, nor levy any general assessments against the membership, except that they may, in individual cases, remit annual dues or assessments.

2. They may in no wise change the Constitution or By-Laws.

3. They may neither elect new members nor alter the status of existing members, other than to apply the provisions of Article IV.—Sec. 2.

4. They may not deplete the principal of the Endowment Fund.

SEC. 2.—Officers and Councilors shall be elected at the annual meeting of the Association, and shall take office on the first day of January, next succeeding. The President and the Vice-President shall be elected for a one year term of office and neither may be reelected to succeed himself in the same office. The Secretary, the Treasurer and the Editor shall be elected for a one year term of office, and any or all may be reelected indefinitely. The outgoing President shall automatically become a Councilor for a one year term of office. The other four Councilors shall be elected, one each year, for a four year term of office, but no Councilor may be reelected to succeed himself.

SEC. 3.—Vacancies occurring among the officers and councilors during the year shall be temporarily filled by action of the Council, subject to approval of the Association at the next regularly convened meeting.

## Article VI—Committees

SEC. 1.—At the opening session of the annual meeting there shall be elected, after nomination from the floor of the Association, a Nominating Committee of three. This Committee shall prepare a slate of nominees for officers and councilors and shall present their report at the executive session of the Association.

SEC. 2.—The Council is empowered to appoint a Membership Committee, an Auditing Committee, a Program Committee, a Necrology Committee and such other committees as may in its opinion be necessary. All such committees shall render their report at the executive session of the Association.

SEC. 3.—The Editor is empowered to appoint an Editorial Board, subject only to the approval of the Council.

SEC. 4.—The Association as a whole may authorize the Council to appoint Scientific or Research Committees for the purpose of investigating thoracic problems and may further authorize the Council to support financially such committees to a limited degree. In appointing such committees, the Council shall be governed by the provisions of the By-Laws.

## Article VII—Finances

SEC. 1.—The fiscal year of the Association shall coincide with the calendar year. The books of the Association shall be kept and audited on this basis.

SEC. 2.—Members shall contribute to the financial maintenance of the Association through the medium of initiation fees, annual dues and special assessments. The amount of the annual dues and the initiation fees shall be determined by the By-Laws.

Special assessments may be charged against individual members, as determined by the By-Laws, when material submitted for publication in the annual transactions of the Association exceeds such limits as may be set in the By-Laws. If, at the end of any fiscal year, there be a deficit in the current funds of the Association, the Council may send out notices to that effect and invite Active Members to contribute the necessary amount so that no deficit be carried over from one fiscal year to another. The Association may, in any regularly convened meeting, vote a special assessment for any purpose whatever, and such special assessment shall become an obligatory charge against the classes of members affected thereby.

SEC. 3.—To meet the current expenses of the Association, there shall be available all revenue derived from annual dues, special assessments, and income from Endowment Fund, subject to the provisions of Section 4 following. Funds derived from the payment of initiation fees shall not be available for current expenses.

SEC. 4.—All funds derived from the payment of initiation fees shall be placed in a special fund, to be invested and reinvested in legal securities, and to be held intact as an Endowment Fund. The Council is responsible for the proper management of the Endownment Fund. The income from this fund may be utilized for such purposes as may be determined in the By-Laws. If at the end of any fiscal year there be a surplus in the current funds of the Association, the Council may divert such surplus into the Endowment Fund, but in no case may the Council divert any of the principal of the Endowment Fund into the current account of the Association.

In the event of the dissolution of the Association, the Endowment Fund shall be distributed among national institutions of the United States and Canada in a proportion equal to the then existing ratio between the numbers of citizens of the two nations who are members of the Association.

### Article VIII—Meetings

SEC. 1.—The time, place, duration and procedure of the annual meeting of the Association shall be determined by the Council, *subject only to the requirements of the Congress of American Physicians and Surgeons* (Amendment to delete this Section passed first reading 1936), and the provisions of the By-Laws.

SEC. 2.—A special meeting of the Association may be called on one month's notice on the written request of fifteen members. The specific purposes of the meeting must be stated in the request and in the official call for the meeting.

SEC. 3.—The annual meeting of the Council shall be held at or near the close of the fiscal year.

### Article IX—Amendments

SEC. 1.—This Constitution shall in no wise be changed except by a three-fourths vote of the members present at an annual meeting, and further provided that the proposed alteration or amendment shall have been moved and seconded at a previous annual meeting, and that printed copies of the suggested alteration or amendment shall have been circulated among the members, and that the members shall have been specifically advised that such alteration or amendment will be voted upon.

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## BY-LAWS

### Article I

SEC. 1.—These By-Laws shall merely interpret the Constitution and specifically apply its principles. They shall set forth no principles not included in the Constitution.

### Article II

SEC. 1.—All papers read before the Association shall become the property of the Association. Authors shall leave original copies of their manuscripts with the Editor, at the time of presentation, for publication in the official journal.

SEC. 2.—When the number of papers makes it desirable, the Council may require authors to present their papers in abstract, and may set a time limit on discussions.

SEC. 3.—Members are urged to cooperate with the Registry for Chest Tumors and all other financially supported Scientific Committees of the Association.

## Article IV

SEC. 1.—Proposals for election to membership in this Association must be made to the Membership Committee. After approval by this Committee and by the Council, the names are to be submitted to the members at an annual meeting of the Association. A three-fourths vote of those present and voting shall be required to elect. Candidates shall be presented in groups in the following order: Candidates for Honorary Membership; retirement of Active Members to Senior Membership; Associate Members eligible to Active Membership; candidates for Active Membership; candidates for Associate Membership.

SEC. 2.—Active Membership shall be limited to 100. The candidate, to be eligible, must be resident in the United States or Canada, must be a graduate in medicine of not less than ten years' standing, and must have made a meritorious contribution to knowledge pertaining to the thoracic field. In unusual circumstances the ten-year requirement will be waived.

SEC. 3.—Associate Membership shall be limited to seventy-five. The candidate, to be eligible, must be a native of or resident in the United States or Canada, and must be a graduate in medicine of not less than five years' standing. The limited period of time for Associate Membership, as required by Article IV, Section 2, of the Constitution, shall be five years.

SEC. 4.—The number of Senior Members shall be unlimited. The candidate, to be eligible, must have been an Active Member of this Association for not less than ten years, or, having been an Active Member, have passed the age of sixty, or become incapacitated by illness.

SEC. 5.—Honorary Members shall be limited to twenty-five. The candidate, to be eligible, may be a citizen of any country, does not necessarily have to hold a degree in medicine, but must have made an exceptional contribution to knowledge pertaining to the thoracic field.

SEC. 6.—Any candidate for membership in this Association who has failed of election for three years shall be automatically dropped from the list of candidates and his name may not be again proposed until after a lapse of five years.

SEC. 7.—The Council may recommend that any member whose dues are in arrears for two years, or who has been absent, without sufficient excuse, from four consecutive annual meetings, shall have his membership withdrawn.

## Article V

SEC. 1.—The President of the Association shall perform all duties customarily pertaining to the office of President. He shall not only preside at all meetings of the Association, but also at all meetings of the Council. The President shall be elected from the Active Members of the Association.

SEC. 2.—The Vice President of the Association shall perform all duties customarily pertaining to the office of the Vice President, not only as to the Association, but also as to the Council. The Vice President shall be elected from the Active Members of the Association.

SEC. 3.—The Secretary of the Association shall perform all duties customarily pertaining to the office of Secretary. He shall serve not only as Secretary of the Association but also as Secretary of the Council. The Secretary shall be elected from the Active Members of the Association.

SEC. 4.—The Treasurer of the Association shall perform all duties pertaining to the office of Treasurer. He shall not only serve as Treasurer of the Association but shall also serve as custodian of the Endowment Fund. The Treasurer shall be elected from the Active Members of the Association.

SEC. 5.—The Editor of the Association shall be the Editor of the official Journal; shall, ex officio, be the Chairman of the Editorial Board; and shall be custodian of the permanent completed archives and records of the Association. The Editor shall be elected from the Active or Senior Members of the Association.

SEC. 6.—The Councilors of the Association shall hold office as specified in the Constitution. They may be elected from either the Active Members or the Senior Members of the Association.

SEC. 7.—In the event of a vacancy occurring in the office of President, the Council shall advance the Vice President to the Presidency and appoint a new Vice President under the provisions of Article V, Section 3, of the Constitution.

## Article VI

SEC. 1.—The Nominating Committee shall consist of three Active or Senior Members elected in accordance with the provisions of Article VI, Section 1, of the Constitution. The Council shall instruct the Committee as to the vacancies which are to be filled by election.

SEC. 2.—The Membership Committee shall consist of five Active or Senior Members appointed in accordance with the provisions of Article VI, Section 2, of the Constitution. The Council may appoint not more than one of its own members to serve on this Committee. The duties of the Membership Committee are to investigate all candidates for membership in the Association and to report their findings as expeditiously as possible to the Council through the Secretary of the Association. This Committee is also charged with searching the literature of this and other countries to the end that proper candidates may be presented to the Association for consideration. Appointment to this Committee shall be for a period of one year, and not more than three of the members may be reappointed to succeed themselves.

SEC. 3.—The Auditing Committee shall consist of three Active or Senior Members appointed in accordance with the provisions of Article VI, Section 2, of the Constitution. None of these may be selected from the officers or councilors of the Association. Their duty shall be to audit the accounts of the Association at the end of each fiscal year, and render their report to the Council of the Association through the Secretary. Appointments to this Committee shall be made for a one-year term. Not more than two members may be reappointed to succeed themselves.

SEC. 4.—The Program Committee shall consist of five members: The President of the Association, the Secretary of the Association, the Editor of the Association and two members at large, one of whom shall be resident at or near the place of annual meeting. The duties of this Committee shall be to arrange, in conformity with instructions from the Council, the scientific program for the annual meeting.

SEC. 5.—The Necrology Committee shall consist of three Active or Senior Members, and shall be appointed in accordance with the provisions of Article VI, Section 2. Appointments of this Committee shall be for a one-year term of office. Not more than two members of this Committee may be reappointed to succeed themselves. The Council may, if it so desires, appoint one of its own members to serve as chairman of the Committee. The duties of the Necrology Committee shall be to prepare suitable resolutions and memorials on the death of any member of the Association.

SEC. 6.—The Editorial Board shall be appointed by the Editor, subject only to the approval of the Council. The Editor shall be, ex officio, the chairman of this board and shall be privileged to appoint and indefinitely reappoint such members of the Association, regardless of class of membership, and such nonmembers of the Association as in his opinion may be best calculated to meet the editorial requirements of the Association.

SEC. 7.—When Scientific or Research Committees are authorized by the Association, the Council shall appoint the Chairmen of these Committees, with power to organize their committees in any way best calculated to accomplish the desired object, subject only to the approval of the Council. Financial aid rendered to such Committees shall not exceed such annual or special appropriations as may be specifically voted for such purposes by the Association as a whole.

Article VII

SEC. 1.—Honorary Members of the Association are exempt from all initiation fees, dues and assessments.

SEC. 2.—Annual dues for Active Members shall be $20.

SEC. 3.—Annual dues for Associate Members shall be $10.

SEC. 4.—Annual dues for Senior Members shall be $10.

SEC. 5.—Initiation fee for those elected directly to Active Membership shall be $15.

SEC. 6.—Initiation fee for those elected to Associate Membership shall be $10. If and when an Associate Member is elected to Active Membership, he shall pay an additional $5 initiation fee.

SEC. 7.—In illustrating the annual transactions the Association will incur expenses not to exceed $15 per article. If the cost for illustrating any article exceeds the sum of $15 the excess shall be charged as a special assessment against the member concerned.

SEC. 8.—Income from the Endowment Fund shall be expended as the Council directs.

Article VIII

SEC. 1.—When the Association convenes for its annual meeting, it shall immediately go into executive session but the business at this session shall be limited to:

1. Election of Nominating Committee.
2. Appointment of necessary committees.
3. Miscellaneous business of an urgent nature.

SEC. 2.—The annual executive session of the Association shall be held at the opening of the afternoon session of the second day of the meeting. The order of business shall be:

1. Reading of the minutes of the preceding meetings of the Association and Council.
2. Report of the Treasurer for the last fiscal year.
3. Report of the Auditing Committee.
4. Report of the Treasurer for the current year to date.
5. Report of the Necrology Committee.
6. Report of the Program Committee.
7. Action on amendments to the Constitution and By-Laws.
8. Action on recommendations emanating from the Council.
9. Unfinished Business.
10. New Business.
11. Report of the Membership Committee.
12. Election of new members.
13. Report of the Nominating Committee.
14. Election of officers.

SEC. 3.—At the annual meeting of the Council the order of business shall be:

1. Reading of minutes.
2. Treasurer's report.
3. Appointment of committees.
4. Determination of instructions to be given to Auditing, Membership, Necrology and other committees except Program Committee.
5. Determination of details in connection with the annual meeting.
6. Appointment and instructions to the Program Committee.
7. Unfinished Business.
8. New Business.

SEC. 4.—There shall be an annual meeting of the Council.

## Article X

SEC. 1.—These By-Laws shall in no wise be changed except by a two-thirds vote of the members present at the annual meeting or a properly convened meeting of the Association, and further provided that the proposed action or amendment shall have been moved and seconded by not less than three of the members in a properly convened annual or special meeting of the Association.

SEC. 2.—These By-Laws may be suspended in whole or in part for a period of not more than twelve hours by a unanimous vote of those present at any regularly convened meeting of the Association.

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